# A STUDY OF
# NON-MALIGNANT LIVER LESIONS


**Dissertation submitted in partial fulfillment of regulation for the award of M.S. Degree in General Surgery**

**(Branch I)**


The Tamilnadu
Dr. M.G.R. Medical University
**Chennai**
**March 2009**

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## Coimbatore Medical College
**Coimbatore - 641 014**

# CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. S. SELVAKUMAR** (28-12-1981) and submitted in partial fulfillment of the requirements for the Degree of M.S., General Surgery, Branch I of The Tamilnadu Dr. M.G.R. Medical University, Chennai

Date : Unit Chief

Date : Professor & Head
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# DECLARATION

I solemnly declare that the dissertation titled **"A Study of Non-Malignant Liver Lesions"** was done by me from July 2006 onwards under the guidance and supervision of Professor **Dr. G. MOHAN M.S.**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery (Branch I).

Place : **Dr. S. SELVAKUMAR** (28-12-1981)

Date :

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## ETHICS COMMITTEE

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| *Course* | : | M.S. (GENERAL SURGERY) |
| *Period of Study* | : | 2006 - 2009 |
| *College* | : | COIMBATORE MEDICAL COLLEGE |
| *Dissertation Topic* | : | A STUDY OF NON-MALIGNANT LIVER LESIONS |

*The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.*

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*Date :* 8.10.2007

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*Ethics Committee*

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# CONTENTS

# INTRODUCTION

Hepatic infections constitute a major portion of Non Malignant Liver lesions. This study is chosen for the following reasons.

- Infective lesions of the liver are most frequently encountered.
- They are amenable for treatment and the cure rates are higher.
- The diagnosis has been rendered easier due to the advances in imaging sciences.
- This study offers an insight to asses these diseases at an early stage and plan successful therapy.

# AIM OF THE STUDY

The aim is to study the following aspect of Non Malignant Liver lesions

- The incidence of Non Malignant Liver lesions among the patients admitted in Surgical wards of Coimbatore Medical College Hospital during the period of July 2006 to August 2008.
- The clinical presentation and management of Infective Non Malignant Liver lesions namely amoebic liver abscess, pyogenic liver abscess and Hydatid cyst.
- Analysis of the different modes of management of the liver abscess which will include

    - Medical management mainly includes administering Metronidazole in amoebic liver abscess and broad spectrum antibiotics in pyogenic abscess, usually third generation cephalosporins and amino glycosides were often administered before microbiological diagnosis and these are modified when sensitivities were known. All the abscesses of less than 5 cm were managed conservatively.
    - Ultra sound guided percutaneous drainage using aspiration needle for collections larger than 5 cm and finally, this was combined with the medical management.
    - Open surgical drainage in cases unresponsive to non operative treatment, in a patient with deteriorating condition in spite of medical management and repeated drainage and for complications like rupture into the peritoneal cavity.

- To review the available literature with particular reference to the clinical presentation, investigations, Complication and methods of treatment of infective non-malignant liver lesions practiced in Coimbatore Medical College Hospital.

# REVIEW OF LITERATURE

## HISTORY

The amoeba, E. histolytica was discovered by Friedrich Losch in 1873 in Russia. In the second century AD, Galen and Celsus both described liver abscesses that were probably amoebic, and the works of Aretaeus, Archigenes, Aurelanus, and Avicenna toward the end of the first millennium give good accounts of both dysentery and hepatic involvement. In the 19th century, several books mainly concerned with diseases in India, including Researches into the Causes, Nature and Treatment of the More Prevalent Diseases of India and of Warm Climates Generally by James Annersley, clearly refer to both intestinal and hepatic amoebiasis. The authoritative report by William Thomas Councilman and Henri Lafleur, working at the Johns Hopkins Hospital in 1891, represents a definitive statement of what was known about the pathology of amoebiasis at the end of the 19th century, and much of it is still valid today [80].

The massive bladder-like hydatid cysts, particularly in the liver, were well known in ancient times, and there are references to such cysts in ritually slaughtered animals in the Babylonian Talmud and, in animals slaughtered for food, by Hippocrates in the fourth century BC, Arataeus in the first century AD, and Galen in the second century AD. It was not until 1853 that Carl von Siebold demonstrated that Echinococcus cysts from sheep gave rise to adult tapeworms when fed to dogs, and in 1863 Bernhard Naunyn found adult tapeworms in dogs fed with hydatid cysts from a human.

# SURGICAL ANATOMY OF THE LIVER

## GENERAL DESCRIPTION AND TOPOGRAPHY

The liver is a solid gastrointestinal organ whose mass (1200 to 1600 g) largely occupies the right upper quadrant of the abdomen. The liver extends superiorly to the height of the fifth rib on the right and the sixth rib on the left, laterally up to 8$^{th}$ in the midaxillary line and posteriorly 9$^{th}$ rib on the right side. The posterior surface straddles the inferior vena cava (IVC). The superior surface of the liver is convex and is molded to the diaphragm, whereas the inferior surface is mildly concave and extends to a sharp anterior border [1, 58].

The liver is invested in Glisson's Capsule except for the gallbladder bed, the porta hepatis, and posteriorly to the right of the IVC (bare area). The diaphragmatic peritoneal duplications are referred to as the coronary ligament whose lateral margins on either side are the right and left triangular ligaments. From the center of the coronary ligament emerges the falciform ligament, which extends anteriorly to the diaphragm, abdominal wall, and umbilicus. The ligamentum teres runs along the inferior edge of the falciform ligament. The falciform ligament was used historically to mark the division of the right and left lobes of the liver. On the posterior surface of the left liver running from the left portal vein in the porta hepatis toward the left hepatic vein and the IVC is the ligamentum venosum, which also runs in a fissure (Figure 1).

## LOBAR ANATOMY

The liver was divided into right and left lobes, determined by portal and hepatic vein branches. Briefly, a plane without any surface markings running from the gallbladder to the left side of the IVC known as the portal fissure or Cantlie's line divided the liver into right and left lobes. The right lobe was further divided into anterior and posterior segments. The left lobe was divided into a medial segment also known as the quadrate lobe that lies to the right of the falciform ligament and a lateral segment lying to the left of it [3].

## FUNCTIONAL ANATOMY

The functional anatomy (Figure 2 & 3) of the liver is composed of eight segments that are each supplied by a single portal triad (pedicle). These segments are further organized into four sectors that are separated by scissurae containing the three main hepatic veins. The four sectors are even further organized into the right and left liver. This system was originally described in 1957 by Goldsmith and Woodburne as well as by Couinaud [2].

The main scissura contains the middle hepatic vein, which divides the liver into right and left hemi livers. The right liver is divided into an anterior (segments V & VIII) and a posterior (segments VI & VII) sector by the right scissura. The left liver is split into an anterior (segments III & IV) and posterior (segment II) sector by the left scissura.

The caudate lobe (segment I) is the dorsal portion of the liver and embraces the IVC on its posterior surface. The main bulk of the caudate lobe is to the left of the IVC, but inferiorly it traverses between the IVC and left

portal triad, where it fuses to the right liver (segments VI and VII). This part is known the caudate process. The left portion of the caudate lobe lies in the lesser omental bursa and is covered anteriorly by the gastrohepatic ligament (lesser omentum).

# BLOOD SUPPLY

## PORTAL VEIN

The portal vein provides about 75% of hepatic blood flow and it provides 50% to 70% of the liver's oxygenation. The portal vein forms behind the neck of the pancreas at the confluence of the superior mesenteric vein and the splenic vein at the height of the second lumbar vertebra. The length of the main portal vein ranges from 5.5 to 8 cm, and its diameter is usually about 1 cm. The portal vein divides into main right and left branches at the hilum of the liver. The left branch of the portal vein runs transversely in the umbilical fissure, where it gives off branches to segments II and III and feeds back branches to segment IV (Figure 2). The left portal vein also gives off posterior branches to the left side of the caudate lobe. The right portal vein has a short extra hepatic course and usually enters the substance of the liver, where it splits into anterior and posterior sectoral branches (Figure 4).

## HEPATIC ARTERY

The hepatic artery provides approximately 25% of the hepatic blood flow and 30% to 50% of its oxygenation. A number of smaller perihepatic arteries derived from the inferior phrenic and the gastroduodenal arteries also supply the liver. These vessels are important sources of collateral blood flow

in the event of occlusion of the main hepatic arterial inflow. The common description of the arterial supply to the liver and biliary tree is only present approximately 60% of the time (Figure 5). The celiac trunk originates directly off the aorta just below the aortic diaphragmatic hiatus and gives off three branches: the splenic artery, the left gastric artery, and the common hepatic artery. The common hepatic artery beyond the take-off of the gastroduodenal is called the proper hepatic artery and divides into right and left branches at the hilum. The left hepatic artery heads vertically toward the umbilical fissure to supply segments I, II, and III. The left hepatic artery usually gives off a middle hepatic artery branch that supplies segment IV. The right hepatic artery usually runs posterior to the common hepatic bile duct and enters Calot's triangle (bordered by the cystic duct, common hepatic duct, and the liver edge), where it gives off the cystic artery to supply the gallbladder. Most often, the hepatic artery originates off of the celiac trunk, but different branches or the entire hepatic arterial system can originate off of the superior mesenteric artery. A replaced or accessory left hepatic artery usually originates from the left gastric artery.

## HEPATIC VEINS

The three major hepatic veins drain from the superior and posterior surface of the liver directly into the IVC (Figure 2&3). The right hepatic vein runs in the right scissura and drains the majority of the right liver after a short (1 cm) extra hepatic course into the right side of the IVC. The left and middle hepatic veins usually join intrahepatically and enter the left side of the IVC as a single vessel, although they may drain separately. The left hepatic vein runs

in the left scissura and drains segments II and III while the middle hepatic vein runs in the portal scissura draining segment IV and some of the anterior sector of the right liver. The umbilical vein runs under the falciform ligament between the left and middle veins, and usually empties into the left hepatic vein. Multiple small venous branches drain posteriorly directly into the IVC. Venous drainage of the caudate lobe is through multiple small hepatic veins that drain directly into the IVC and a large tributary that drains superiorly into the left hepatic vein.

## BILIARY SYSTEM

The intrahepatic bile ducts are terminal branches of the main right and left hepatic ductal branches that invaginate Glisson's capsule at the hilum along with the corresponding portal vein and hepatic artery branches forming the peritoneal covered portal triads (Figure 3). Along these intrahepatic portal triads, the bile duct branches are usually superior to the portal vein while the hepatic artery branches run inferiorly. The left hepatic duct drains segments II, III, and IV and it joins the right hepatic duct at the hilum. The right hepatic duct drains the right liver and is formed by the joining of the anterior sectoral duct and the posterior sectoral duct. The short right hepatic duct meets the longer left hepatic duct forming the confluence anterior to the right portal vein, constituting the common hepatic duct. The caudate lobe has its own biliary drainage, which is usually through both right and left systems, although in up to 15% of cases drainage is through the left system only and in 5% it is through the right system only.

## NERVES

Sympathetic fibers originating from T7 through T10 innervates the hepatic arteries, the gallbladder and extra hepatic bile ducts. Parasympathetic fibers from both vagal nerves innervate the gallbladder and extra hepatic bile ducts.

## LYMPHATICS

The majority of lymph node drainage from the liver is to the hepatoduodenal ligament. From here lymphatic drainage usually continues along the hepatic artery to the celiac lymph nodes and from here to the cisterna chyli. Lymphatic drainage can also follow the hepatic veins to lymph nodes in the area of the suprahepatic IVC and through the diaphragmatic hiatus.

## MICROSCOPIC ANATOMY

The organization of hepatic parenchyma is into microscopic functional units as an acinus or a lobule (Figure 6). A lobule is made up of a central terminal hepatic venule surrounded by four to six terminal portal triads forming a polygonal unit. This unit is lined on its periphery by terminal portal triad branches. In between the terminal portal triads and the central hepatic venule, hepatocytes are arranged in plates, one cell thick, surrounded on each side by endothelial-lined and blood-filled sinusoids (Figure 7&8). Blood flows from the terminal portal triad through the sinusoids into the terminal hepatic venule. Bile is formed in the hepatocytes and emptied into terminal canaliculi that form on the lateral walls of the intercellular hepatocyte, ultimately coalescing into

bile ducts and flowing toward the portal triads. This functional hepatic unit provides a structural basis for the many metabolic and secretory functions of the liver. Between the terminal portal triad and the central hepatic venule there are three zones. Zones 1 through 3 fans out from the terminal portal triad toward the central hepatic venule. Zone 1, known as the periportal zone, is exposed to an environment rich in nutrients and oxygen. Zones 2 (intermediate zone) and 3 (perivenular zone) are exposed to environments less rich in oxygen and nutrients. The cells of the different zones respond differently to toxin exposure as well as hypoxia. This anatomic arrangement also explains the phenomenon of centrilobular necrosis from hypotension, with zone 3 being the most susceptible to decreases in oxygen delivery.

## LIVER FUNCTIONS

The liver consists of four physiologic-anatomic units that are interrelated:

- The circulatory system. A dual blood supply nourishes the liver and acts as a vehicle for material absorbed from the intestinal tract to be utilized in the metabolic pool.

- Biliary passages. These serve as channels of exit for materials secreted by liver cells, including bilirubin, cholesterol, and detoxified drugs. This system originates with the Golgi apparatus adjacent to the microvilli of the bile canaliculi and eventually terminates in the common bile duct.

- The reticuloendothelial system. This system has 60 percent of its cellular elements in the liver and includes the phagocytic Kupffer cells and endothelial cells.

- The functioning liver cells (hepatocytes), which are capable of wide variation of activity. The metabolic pool in the liver serves the needs of the entire body. The cell performs both anabolic and catabolic activities, secretes, and stores. The large amount of energies required for these transformations result from the conversion ATP to ADP. A second source is the aerobic oxygenation in the metabolic pool via the tricarboxylic acid cycle of Krebs [60].

# ETIOLOGY OF LIVER ABSCESS

The liver is probably exposed to portal venous bacterial loads on a regular basis and clears this bacterial load without problems in the usual circumstance. The development of a hepatic abscess occurs when the inoculum of bacteria, regardless of the route of exposure, exceeds the livers ability to clear it. This results in tissue invasion, neutrophil infiltration, and the formation of an organized abscess.

The potential routes of hepatic exposure to bacteria are

- Biliary tree,
- Portal vein,
- Hepatic artery,
- Direct extension of a nearby focus of infection, and
- Trauma.

**Amoebic liver abscess** (80% of liver abscess in this study) is the most common inflammatory space-occupying lesion of the liver. The causative agent is a protozoan, Entamoeba histolytica. Ten percent of the world population harbors E. histolytica in their colon, 10% of them may develop invasive amoebiasis and 1 to 10% of the patients develop amebic abscess in the liver[56].

Hepatic infections from the biliary tree are next common identifiable cause of hepatic abscess. **Biliary obstruction** results in bile stasis, with the

potential for subsequent bacterial colonization, infection, and ascension into the liver. This process is known as ascending suppurative cholangitis. The nature of biliary obstruction is mostly related to stone disease or malignancy[18].

**The portal venous system** drains the gastrointestinal tract, and therefore any infectious disorder of the gastrointestinal tract can result in an ascending portal vein infection (pyelophlebitis) with exposure of the liver to large amounts of bacteria. The most common causes of pyelophlebitis are diverticulitis, appendicitis, pancreatitis, inflammatory bowel disease, pelvic inflammatory disease, perforated viscus, or omphalitis in the newborn. Hepatic abscess has also been associated with colorectal malignancy [4, 5, 6].

Any **systemic infection** (e.g., endocarditis, pneumonia, osteomyelitis) can result in bacteremia and infection of the liver via the hepatic artery. Hepatic abscess from systemic infections may also reflect an altered immune response, such as in patients with malignancy, acquired immune deficiency syndrome, or disorders of granulocyte function. Children with chronic granulomatous disease are particularly susceptible.

Hepatic abscess can be the result of **direct extension** of an infective process. Common examples of this include suppurative cholecystitis, subphrenic abscess, perinephric abscess, or even perforation of the intestine directly into the liver.

**Penetrating and blunt trauma** can result in an intrahepatic hematoma or an area of necrotic liver that can subsequently develop into an abscess. Bacteria may have been introduced from the trauma, or the affected area may be seeded from systemic bacteremia. Other mechanisms of iatrogenic hepatic necrosis such as hepatic artery embolization or, more recently, thermal ablative procedures can be complicated by abscess.

**Cryptogenic abscesses** (20%) predominate in many series and are more common in recent case series. Possible explanations for cryptogenic hepatic abscess are undiagnosed abdominal pathology, resolved infective process at the time of presentation, or host factors such as diabetes or malignancy rendering the liver more susceptible to transient hepatic artery or portal vein bacteremias.

# PATHOGENESIS OF PYOGENIC LIVER ABSCESS

The etiology of liver abscess helps us to predict the size, number and location of abscess in the liver. Portal, cryptogenic and traumatic hepatic abscess are large and solitary. Biliary and arterial abscess are multiple and small. Total biliary obstruction is associated with elevated pressure within the biliary tree, an acute septic course, and miliary microabscesses throughout the hepatic parenchyma—a process that has been termed ‘acute suppurative cholangitis'. Infection associated with less complete obstruction is associated with normal biliary tract pressure, a subacute course, and macroscopic abscesses [7].

In the review by Gyurffy and colleagues 40% of pyogenic hepatic abscess were found to be 1.5 to 5cm in diameter, 40% were 5 to 8cm and 20% were 8cm or larger. Overall, 68% of liver abscess localize in the right lobe and most of them solitary. Abscess in left lobe of liver occur in 12% of cases. 20% of patients have liver abscess in both lobes. Liver abscess of biliary etiology are multiple and bilateral. A bilateral disease occurs in 90% of cases with a biliary and arterial source of infection. Fungal hepatic abscess are most often multiple, bilateral and disseminated.

Table 1 explains the pathogenesis of pyogenic liver abscess by etiology.

## TABLE 1

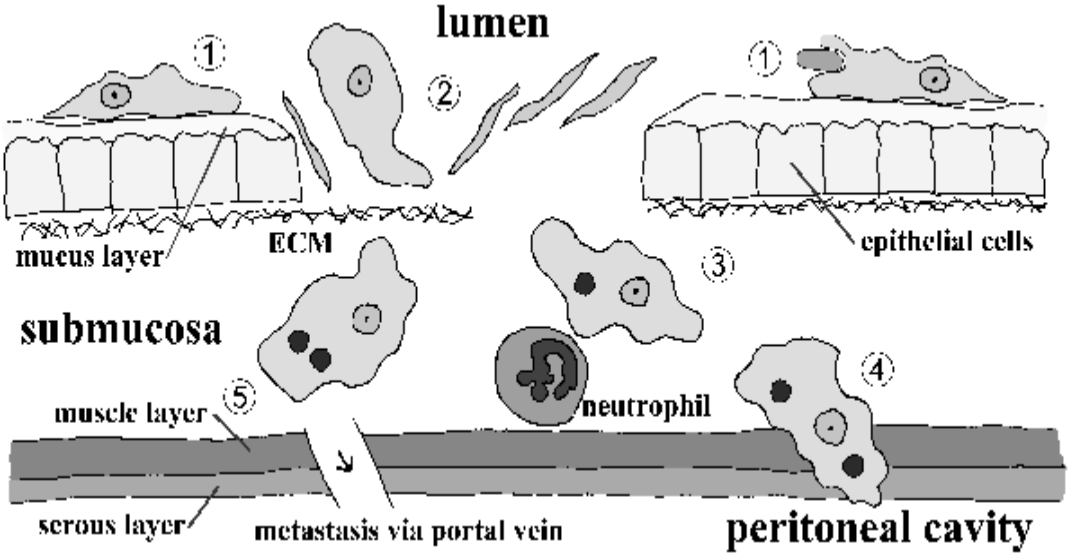
| Etiology | Source of infection | Distribution | Primary microorganism |
|---|---|---|---|
| Biliary system | Cholangitis, biliary obstruction | Both lobes, multiple | Polymicrobial, gram negative aerobes and anaerobes eg.E.coli |
| Portal circulations | Intra-abdominal infections | Right > Left Multiple or single | Polymicrobial, enteric aerobes and anaerobes E-faecalis, B fragilis |
| Arterial circulation | Bactereimia, systemic infection | Both lobes, multiple | Single species, gram positive aerobes like Staph-aureus and Strep-pyrogenes |
| Trauma | Direct exposure, necrosis | Area of injury | Single species, gram(+) aerobes, Staph-aureus and Strep- pyogenes |
| Direct extension | Cholecystitis and perforated ulcer | Adjacent area | Single species, gram (-) aerobes, E-coli |
| Cryptogenic | Unknown | Rt > Lt | Single species, anaerobic B. fragilis |

# PATHOGENESIS OF AMOEBIC LIVER ABSCESS

E. histolytica is a protozoan and exists as a trophozoite or as a cyst. E. histolytica complex is made up of two distinct species: E. histolytica, which is capable of invading the colonic mucosa and causing extraintestinal disease; and E. dispar, which remains a gut commensal.

Ingestion of E. histolytica cysts through a fecal-oral route is the cause of amebiasis. Humans are the principal host, and the main source of infection is human contact with a cyst-passing carrier. Contaminated water and vegetables are also a route of human infection. Once ingested, the cysts are not degraded in the stomach and pass to the intestines, where the trophozoite is released and passed on to the colon. In the colon, the trophozoite can invade mucosa and result in disease. It is believed that the trophozoites reach the liver through the portal venous system.

E. histolytica trophozoites have the capacity to lyse tissues through a complex set of events, including cell adherence, cell activation, and subsequent release of multiple enzymes resulting in necrosis. The major mechanism is probably enzymatic cellular hydrolysis. Amebic liver abscesses are thus formed by progressing, localized hepatic necrosis, resulting in a cavity containing acellular proteinaceous debris surrounded by a rim of invasive amebic trophozoites[10].




Step 1 - Trophozoites adhere to the mucus layer. This adherence is simply a mechanism for the ameba to crawl along the substratum.

Step 2 - Epithelial cells are killed in a contact dependent manner leading to a disruption of the intestinal mucosa.

Step 3 - The trophozoites will continue to kill host cells in the submucosa.

Step 4 - Disruption of the intestinal wall.

Step 5 - metastasis via the circulatory system.

Adherence, cytotoxicity, and disruption of the tissues are important factors in the pathogenesis of E. histolytica. Parasite proteins which could play a role in these processes include: the **Eh-lectin, amebapore, and proteases** [19].

**Eh-lectin** Adherence of E. histolytica trophozoites to host cells and colonic mucins is mediated by a lectin-activity expressed on the ameba's surface. This lectin binds galactose or N-acetyl-D-galactosamine (GalNAc) with a high affinity and is also called the galactose-inhibitable adherence protein (GIAP) or the Gal/GalNAc lectin. This is an important virulence factor. Vaccine against this is under trail.

**Ameba pore** A family of pore-forming polypeptides has been identified in E. histolytica and E. dispar. The three family members are designated as amebapore A, B and C with amebapore A being predominant expressed. Three of these dimers then assemble into a hollow ring-shaped structure. This hexamer then can intercalate into membranes and introduce 2 nm pores (i.e., holes) which results in cell death.

**Proteases** are enzymes that degrade other proteins and could contribute to the pathogenesis cause by E. histolytica. In this regard, E. histolytica expresses and secretes higher levels of cysteine proteases. This degradeds the mucus and the degraded mucus are less efficient at blocking adherence of trophozoites to epithelial cells. Destruction of the extracellular matrix (ECM) by proteases may also facilitate trophozoite invasion.

# PATHOLOGY OF LIVER ABSCESS

The majority of Pyogenic liver abscesses involve the right lobe of the liver, accounting for three fourths of cases. The explanation for this is not known, but preferential laminar blood flow to the right side has been postulated. The left lobe is involved about 20% of the time, and the caudate lobe is uncommonly involved (5%). Bilobar involvement with multiple abscesses is uncommon. About half of hepatic abscesses are solitary.

Approximately 80 per cent of amebic liver abscesses are solitary and 83 per cent are located in the right lobe of the liver, characteristically high in the dome subjacent to the diaphragm. The propensity for this site reflects the fact that venous return from the right side of the colon into the portal vein is predominantly delivered to the right lobe of the liver. Hepatic amoebic abscess is essentially the result of liquefaction necrosis of the liver, producing a cavity full of blood and liquefied liver tissue (Figure 9). The appearance of this fluid is typically described as “anchovy sauce,” (Figure 10). Microscopically, three zones are recognized: a necrotic center, a middle zone with destruction of parenchymal cells, and an outer zone of relatively normal hepatic tissue in which amebas may be demonstrated. The progressive hepatic necrosis continues until Glisson’s capsule is reached because the capsule is resistant to hydrolysis by the amoebae, and thus amoebic abscesses tend to abut the liver capsule. Bile is lethal to amoebas; thus, infection of the gallbladder and bile ducts does not occur[8, 9].

# MICROBIOLOGY OF LIVER ABSCESS

The heterogeneity of the routes of infection in Pyogenic abscess makes the microbiology variable. Abscesses from pyelophlebitis or cholangitis tend to be polymicrobial, with a high preponderance of gram-negative rods. Systemic infections usually cause infection with a single organism. Many hepatic abscesses are polymicrobial and account for about 40% of cases. Anaerobic organisms are involved 40% to 50% of the time. Escherichia coli is the most common facultative organism isolated from liver abscesses, with Klebsiella pneumoniae being the second most frequent isolate in this category. Bacteroides fragilis is the most common anaerobe isolated. Other common organisms encountered are Staphylococcus aureus, Enterococcus species, Streptococcus viridans. Uncommonly encountered organisms (<10% of cultures) include Pseudomonas, Proteus, Enterobacter, Citrobacter, Serratia, β-hemolytic streptococci, microaerophilic streptococci, Fusobacterium, Clostridium, and other rare anaerobes. Blood cultures are positive in 50% to 60% of cases. Fungal (C. albicans and C. tropicalis) and mycobacterial hepatic abscesses are rare and are almost always associated with immunosuppression [56].

Cultures of amebic abscess are usually negative (sterile abscess). When secondary infection supervenes pus culture become positive.

# CLINICAL FEATURES OF LIVER ABSCESS

The classic description of the presenting symptoms of pyogenic abscess are fever, jaundice, and right upper quadrant pain and tenderness. Unfortunately, this presentation is present only 10% of the time. Fever is present in 90 per cent cases, nausea, vomiting, and abdominal pain occurs in 50 to 75 per cent. Abdominal tenderness in the right upper quadrant is the most common physical finding, being demonstrable in 50 to 75 per cent of affected individuals. Hepatomegaly is demonstrable in approximately 50 per cent of patients with macroscopic liver abscesses. Jaundice is uncommon, unless biliary obstruction is present. Clinical presentation in this study is tabulated in table 4.

Amebic liver abscess has a more subacute presentation than that of pyogenic liver abscess. Most cases have variable clinical features & atypical presentations (Azhar Jawaid Bukhari et al, 2003) Initial symptoms are non-specific: fever, anorexia, night sweats, malaise, nausea and vomiting, and weight loss. As the disease becomes established, right upper quadrant abdominal pain becomes a dominating symptom in at least two-thirds of these patients. Synchronous hepatic abscess is found in one third of patients with active amebic colitis. Approximately 25 per cent of patients exhibit thoracic symptoms. Uncommonly, patients may have a more fulminant presentation, suggesting an acute abdominal surgical emergency. Symptoms and tenderness may be epigastric or left sided if the abscess is located in the left liver[57, 58, 59, 60].

# INVESTIGATIONS

## Haematology

Hemoglobin percentage, total count, differential count, erythrocyte sedimentation rate, clotting time and bleeding time was done for all cases. In pyogenic abscesses, leukocytosis (70% to 90%), elevated erythrocyte sedimentation rate (90 %), and anemia was encountered. In amebic abscess, mild to moderate leukocytosis without eosinophilia and anemia was found.

## Motion Examination

Motion examination was done in all cases of liver abscess. Wet saline preparation, iodine preparation were examined under high power for evidence of trophozoites and cyst. Concentration method was also used in few cases. In greater than 70% of patients with amebic liver abscess stool examination was negative.

## Liver Function Test

Serum bilirubin, SGOT, SGPT, ALP, Total protein, and albumin globulin ratio were done in all cases of liver abscess. Abnormalities of LFTs are generally present. ALP is mildly elevated in 80% of cases, whereas total bilirubin is elevated 20% to 50% of the time. Transaminases are mildly elevated about 60% of the time. The most common LFT abnormality in amoebic liver abscess is an elevated prothrombin time.

## Blood Culture

Blood cultures are positive in 50% to 60% of cases of pyogenic liver abscess. But it is usually negative in amoebiasis.

## Pus Culture

Pus culture was done for all the cases of liver abscess. More than 90% showed organisms in pus culture. Amoebic and parasitic organisms are difficult to identify with both standard or selective staining and culture methods. So these infections should be suspected if bacterial cultures are negative (Sterile abscess).

## Serological tests

The most useful laboratory evaluation is the measurement of circulating antiamebic antibodies that are present in 90% to 95% of patients. An indirect hemagglutinin test has a sensitivity of 90%. This test has largely been replaced by enzyme immunoassays (EIA). The EIA and agar gel diffusion assays has a reported sensitivity of 99% and specificity greater than 90% in patients with hepatic abscess. Unfortunately, the presence of antibodies may reflect old infection and interpretation can be difficult in endemic areas. Levels of antibodies detectable by counterimmunoelectrophoresis and indirect immunofluorescence usually become undetectable within 6 months of acute infection; they may be more useful in evaluating patients in endemic areas. These tests were not done in this study.

## Radiology

The most essential element to making the diagnosis of hepatic abscess is radiographic imaging studies. Chest radiographs, abdominal X-rays was taken in all the cases. Chest x-rays are abnormal in about 50% of the time, and findings generally reflect subdiaphragmatic pathology such as an elevated right hemidiaphragm (Figure 11), right pleural effusion, or atelectasis. Occasionally, these can be left-sided findings in the case of an abscess involving the left liver. Plain abdominal radiographs, in rare cases, can be helpful. They can show air-fluid levels or portal venous gas.

## Ultrasonogram

In pyogenic liver abscess ultrasound usually demonstrates a round or oval area that is less echogenic than the liver and capable of delineating liver lesions as small as 2 cm in diameter. The limitations of ultrasound are it's in ability to visualize lesions high up in the dome of the liver, multiple microscopic abscesses and the fact that it is a user-dependent modality. The sensitivity of ultrasound in diagnosing hepatic abscess is 80% to 95%.

**Modified N'Gbesso classification [76] for Amoebic Abscess of Liver** (G.A. Nari et al, 2008)

| Echo-anatomic forms | Ultrasound characteristics |
|---|---|
| Non-collected AAL (type I) | Heterogeneous hypo-echogenicity<br>Imprecise policyclic limits<br>Dense echogenic outlines<br>Posterior reinforcement of echos<br>Content in echogenic basin |
| Collected AAL (type II) | Heterogeneous hypo-echogenicity<br>Net, regular limits<br>Fine outlines<br>If content is homogeneous in "quicksand",<br>If anechogenic ultrasound type "pseudo-cystic"<br>Or with a "setting sun" level<br>Posterior reinforcement of echoes |
| Healed AAL (type III) | Total healing: Restored ad integrum<br>Persistent or sequela: Cystic or shelled,<br>In ornament, or calcified block. |
| Intermediate | Confluent micro-abscesses |
| AAL with alert signs | Greater than 10 cm, superficial, and thin wall |

In amoebic liver abscess ultrasound has a reported accuracy of approximately 90% when combined with a typical history and clinical presentation. Typical findings on abdominal ultrasound are a rounded lesion abutting the liver capsule without significant rim echoes interpreted as an abscess wall. The contents of the cavity are usually hypoechoic and nonhomogeneous (Figure 12). These findings on ultrasound appear in 40% to 70% of cases. USG was done in all cases.

## Computed Tomography

CT demonstrates similar findings to ultrasound, and lesions are of lower attenuation than surrounding hepatic parenchyma (Figure 13A). High-quality CT can detect intrahepatic collections as small as 0.5 cm in diameter and can be particularly useful in identifying multiple small abscesses or abscesses located near the hemidiaphragm. The pyogenic abscess wall usually shows an intense enhancement on contrast enhanced CT (Figure 13B) which is secondary to increased capillary permeability in the surrounding liver parenchyma (the "double target" sign). The sensitivity of CT in diagnosing pyogenic abscess is 95% to 100%. Magnetic resonance imaging (MRI) can be very helpful in distinguishing the etiology of many hepatic masses but does not appear to have any distinct advantage over CT in diagnosing liver abscess. Abdominal CT is probably more sensitive than ultrasound and is helpful in differentiating amebic from pyogenic abscess, with rim enhancement noted in the latter (Figure 13B & 14). CT scan was done in few cases

## Nuclear medicine studies

Nuclear medicine studies such as gallium scanning or technetium-99m liver scans can be helpful in differentiating pyogenic from amebic abscesses because the latter typically do not contain leukocytes and therefore do not light up on these scans. Because of the nonavailability of this specialty these were not done.

## Differential Diagnosis

Differentiating pyogenic abscess from other cystic infective diseases of the liver such as amebic abscess or echinococcal cyst is important because of differences in treatment. Fortunately, echinococcal cysts can usually be diagnosed by history and characteristic radiologic findings. The presentations of amebic and pyogenic abscess, however, are nearly identical, with some notable exceptions that are critical in distinguishing the two (Table 2). Occasionally, differentiating the two is not possible and diagnostic aspiration or a trial of antiamebic antibiotics may be necessary. Unfortunately, aspiration is only diagnostic in amebic abscess 10% to 20% of the time [11,36].

## Complications [82]

- Rupture into the lungs or pleura causes empyema, hepato-bronchial fistula or pulmonary abscess.
- Rupture into the pericardium is a complication of left lobe liver abscess.
- Intra-peritoneal rupture results in acute peritonitis.
- Left lobe abscess may perforate into the lesser sac.
- Perforation into the portal vein, bile duct or gastrointestinal tract is rare.
- Secondary infections in amoebic liver abscess.

## TABLE 2

| Clinical Features | Amebic Abscess | Pyogenic Abscess |
|---|---|---|
| **Age (yr)** | 20–40 | > 50 |
| **Male: female ratio** | ≥10 : 1 | 1.5 : 1 |
| Solitary vs. multiple | Solitary ≥80% | Solitary 50% |
| Location | Usually right liver | Usually right liver |
| **Travel in endemic area** | Yes | No |
| **Diabetes** | Uncommon (approx. 2%) | More common (approx. 27%) |
| Alcohol use | Yes | Yes |
| Jaundice | Rare | Common |
| Elevated bilirubin | Uncommon | Common |
| Elevated alkaline phosphatase | Common | Common |
| **Positive blood culture** | No | Common |
| **Positive amebic serology** | Yes | No |

## Outcome

Mortality from pyogenic hepatic abscess has been reported from 10% to 20%. In this study there was no mortality. The presence of malignancy, signs of chronic disease such as hypoalbuminemia, signs of severe infection such as marked leukocytosis, Acute Physiology and Chronic Health Evaluation (APACHE) II scores, abscess rupture, bacteremia, and shock are also associated with poor outcome. (Wai-Man Wong et al, 2002)

The mortality for all patients with amebic liver abscess is 2% to 4%. When an abscess ruptures the mortality is reported to be from 6% to as high as 50%. In this study there was no mortality. Factors independently associated with poor outcome are elevated serum bilirubin (>3.5 mg/dL), encephalopathy, hypoalbuminemia (<2.0 g/dL), multiple abscess cavities, abscess volume greater than 500 mL, anemia, and diabetes (M.P.Sharma et al, 2003). Although clinical improvement after adequate treatment with antiamebic agents is the rule, radiologic resolution of the abscess cavity is usually delayed. The average time to radiologic resolution is 3 to 9 months and can take as long as years in some patients [37].

# TREATMENT OF LIVER ABSCESS

Once the diagnosis of pyogenic hepatic abscess is suspected, **broad-spectrum intravenous antibiotics** was started immediately to control ongoing bacteremia and its associated complications. Blood cultures and cultures of the abscess from aspiration were sent for aerobic and anaerobic cultures. Until cultures have specifically identified the offending organism(s), broad-spectrum antibiotics covering gram-negative and gram-positive organisms and anaerobes was used. Combinations of a Cephalosporin, an aminoglycoside, and metronidazole were started. Intravenous antibiotics was administered for 14 days and then replaced with oral preparations to complete a 6-week course. Defervescence occurs within the first week of intravenous antibiotics in most patients with pyogenic hepatic abscesses.

Over the past 20 years, **percutaneous catheter drainage** has become the treatment for most patients of pyogenic liver abscess. Success rates range from 69% to 90%. Percutaneous drainage is carried out under CT or ultrasound guidance, with the insertion of a pigtail catheter using the Seldinger technique. Samples are then withdrawn for microbiologic examination, the abscess cavity is gently irrigated with saline, and the catheter is left in place to provide continuing drainage. Failure to achieve drainage may be due to poor catheter placement, the presence of a multiloculated abscess, excessive viscosity of the abscess contents causing plugging of the drainage catheters, thick abscess walls that do not collapse with drainage, and inadequate anatomic localization of the abscess. Follow-up

ultrasonography or CT scanning is necessary to ensure complete resolution of the process. Relative contraindications to percutaneous catheter drainage include the presence of ascites, coagulopathy, or proximity to vital structures[12, 13, 22]. This procedure is not followed in our institution.

**Percutaneous aspirations** without the placement of a drain have a success rate of 60% to 90% and are somewhat similar to percutaneous catheter drainage. The majority of aspirations, however, require more than one aspiration and one fourth of patients require three or more aspirations. In general, aspiration is recommended for diagnostic uncertainty, for failure to respond to metronidazole therapy in 3 to 5 days, or in abscesses believed to be at high risk for rupture. Abscesses larger than 5 cm in diameter (Rustam Khan et al, 2008) are believed to be at high risk of rupture, and, in particular, when they are located in the left lobe with risk of pericardial rupture therapeutic aspiration is recommended [15, 16, 17]. (Table 3).

This procedure was most commonly performed for treatment of liver abscess in our institution (Figure 15 to 18). In this study about 35 cases (55%) were cured with single aspiration and about 17 cases (27%) were cured with two to three aspirations. There was no mortality following aspiration in this study.

**Surgical exploration** is advised for complication like intra-peritoneal rupture, unstable patients exhibiting signs of continued sepsis despite attempted nonsurgical treatment, and for stable patients who have fevers that

persist for longer than 2 weeks after percutaneous catheter drainage and the institution of appropriate antibiotics (Arshad Zafar et al, 2002). In this study laparotomy was done for three cases (4%) with complication.

Liver resection is occasionally required for hepatic abscess. This may be required for an infected hepatic malignancy, hepatolithiasis, or intrahepatic biliary stricture.

The mainstay of treatment for suspected amoebic abscesses are metronidazole (Blessman et al, 2003), 750 mg oral or 500 mg IV route three times per day for 10 days; clinical improvement (defervescence and decreased abdominal pain) is usually seen within 3-4 days. Other nitroimidazoles (secnidazole, tinidazole 2 g orally once for 3 days) are also as effective. Treatment in the non-responders is with dihydroemetine (1 to 1.5 mg/kg per day, maximum dose 90 mg/day, intramuscularly for 5 days) plus chloroquine phosphate (600 mg base/day for 2 days, followed by 300 mg of chloroquine base orally daily for 2 to 3 weeks).After treatment of the liver abscess, it is recommended that luminal agents are administered to treat the carrier state. Luminal agents effective for amoebiasis include iodoquinol, paramomycin, and diloxanide furoate. Iodoquinol 650 mg orally, three times per day, for 20 days; diloxanide furoate 500 mg orally, three times per day, for 10 days; or paramomycin (250-mg tablets), 500 mg tid for 10 days. In this study, about 9 cases (14%) responded to antibiotic alone.

Rupture into the pleural space usually results in a large and rapidly accumulated effusion that collapses the involved lung. Treatment consists of thoracentesis, but if secondary bacterial infection ensues, more aggressive surgical approaches may be necessary [23]. In this study one case presented with rupture into the pleural space (1%).Rupture can occur into the bronchi and is usually self-limited with postural drainage and bronchodilators. A hepatobronchial fistula may cause cough productive of large amounts of necrotic material that may contain amoebas. This dramatic complication carries a good prognosis. Rarely, a left-sided abscess may rupture into the pericardium and can present as an asymptomatic pericardial effusion or even tamponade. This must be treated with aspiration.

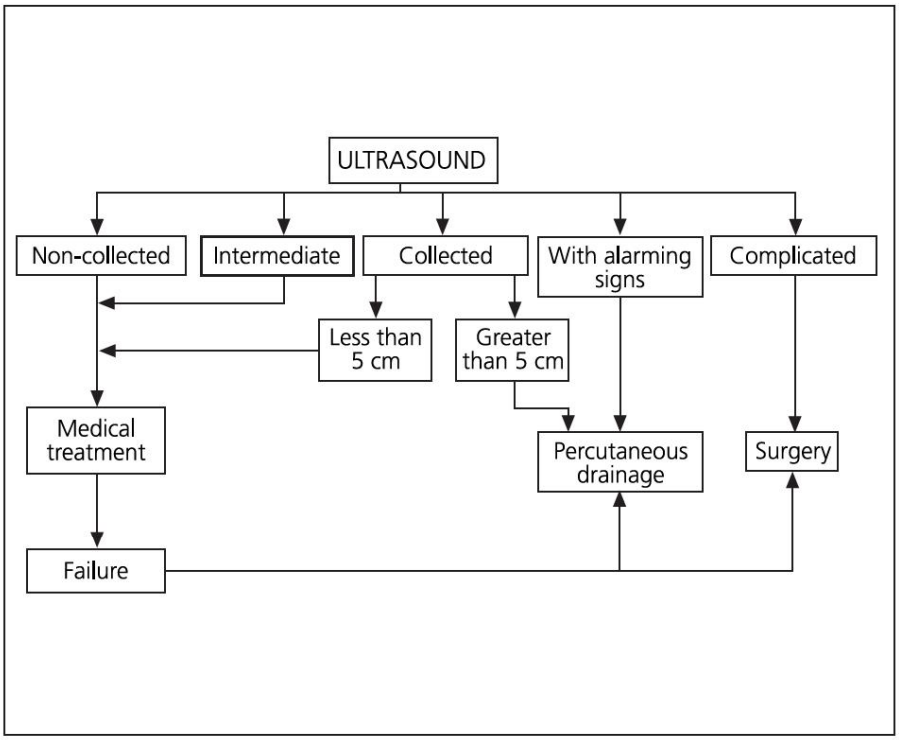
## TABLE 3

(Dela Rey Nel et al, 1989)

| INDICATION FOR ASPIRATION IN A AMOEBIC LIVER ABSCESS |
|---|
| No improvement clinically after 48 - 72 hrs of Chemotherapy |
| Abscesses causing marked tenderness or severe pain |
| All large abscesses (> 5 cm in any dimension) |
| Superficial abscesses (anterior and inferior if > 5 cm in Diameter) |
| Marked elevation of the diaphragm (adjacent to the right dome of diaphragm) |
| Left lobe abscesses (left lobe adjacent to the diaphragm or closer than 2 cm) |
| Negative Serology |

# MANAGEMENT PROTOCOL FOR AMOEBIC LIVER ABSCESS

(G.A.Nari et al, 2008)

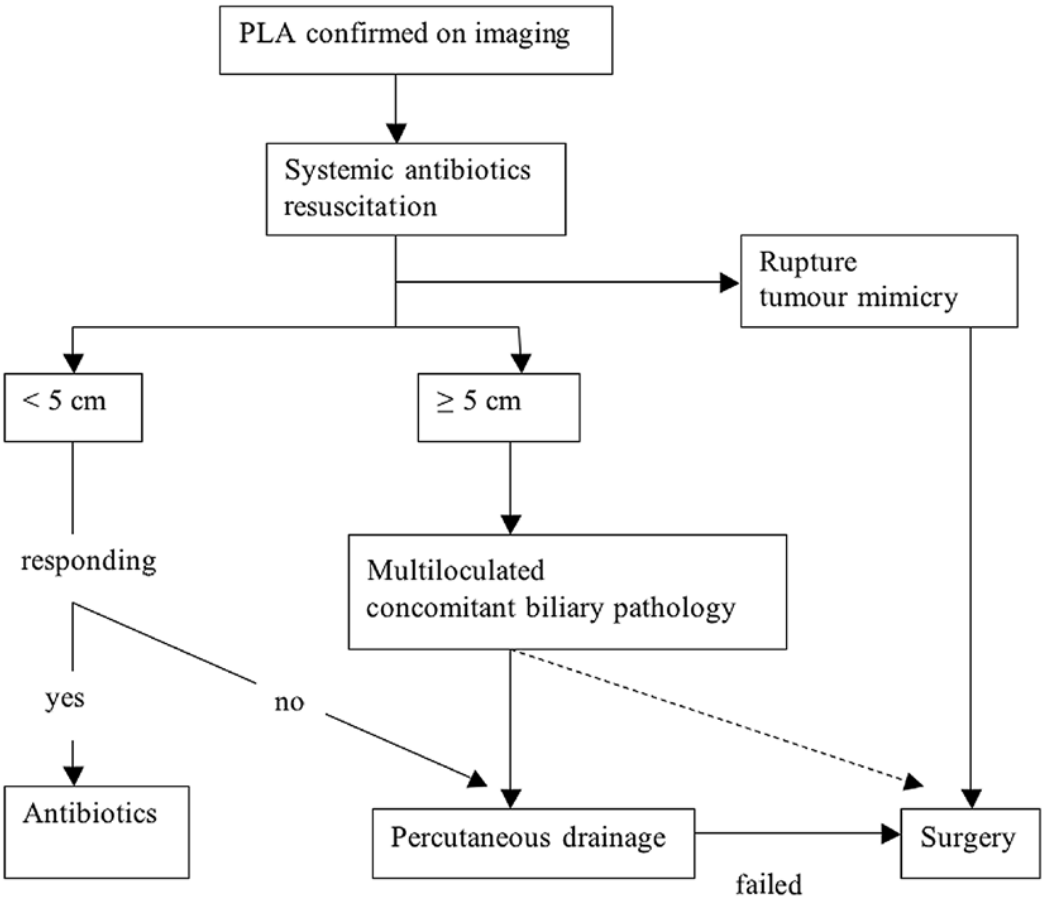



## Prevention of Amoebiasis

Amebic infection is spread by ingestion of food or water contaminated with cysts. Since an asymptomatic carrier may excrete up to 15 million cysts per day, prevention of infection requires adequate sanitation and eradication of cyst carriage. In high-risk areas, infection can be minimized by the avoidance of unpeeled fruits and vegetables and the use of bottled water. Because cysts are resistant to readily attainable levels of chlorine, disinfection by iodination is recommended. There is no effective prophylaxis.

# MANAGEMENT PROTOCOL FOR PYOGENIC LIVER ABSCESS

(Chung Y.F.A et al, 2007)



## EPIDEMIOLOGY OF HYDATID DISEASE LIVER

Hydatid disease is a parasitic infection caused by the larval stage of the cestode Echinococcus granulosus. Hydatid disease or echinococcosis is a zoonosis that occurs primarily in sheep-grazing areas of the world but is common worldwide because the dog is a definitive host. Echinococcosis is endemic in Mediterranean countries, the Middle East, the Far East, South America, Australia, New Zealand, and East Africa. Humans contract the disease from dogs, and there is no human-to-human transmission [26].

## PATHOGENESIS OF HYDATID DISEASE LIVER

There are four species of Echinococcus that cause hydatid disease. E. granulosus is the most common whereas E. multilocularis (E. alveolaris, E. sibiricensis), E. oligartus and E. vogeli account for a small number of cases.

The adult parasite is a 5-mm tapeworm, E. granulosus, which lives in the small intestine of dogs and other canines. The worm has a pyriform head (scolex), a short neck, and only a few segments (proglottids), of which the terminal one releases the ova, 300 to 500 in number. These ova are passed in the faeces and remain viable on the ground for many months. Contaminated grass is ingested by sheep and cattle, and humans may become infected by consuming low growing fruits and vegetables or by handling dogs carrying eggs in hair, saliva, and the anal region. In the jejunum of these intermediate hosts, the larvae (oncospheres) hatch from the eggs, facilitated by digestive enzymes in the duodenum, and migrate through the

bowel wall. They end up in mesenteric lymphatics and in venules of the portal circulation, and are then carried to the liver where they are trapped in the sinusoids and develop into hydatid cysts. Some larvae are not filtered in the liver, but remain in the blood to reach the next station, the lungs. In addition, some may pass through the pulmonary circulation and travel to other organs. Larvae transported in the mesenteric lymphatics are carried to the cisterna chyli, the thoracic duct, and into the general circulation, ending up in a variety of distant sites [27].

When affected sheep and cattle are slaughtered, the viscera and meat may be thrown away and eaten by dogs. The cycle is then closed with the ingested scolices being transformed into adult tapeworms to live again in the canine's small intestine and produce ova.

## PATHOLOGY OF HYDATID DISEASE LIVER

In infection by E. granulosus, 70 per cent of all hydatid cysts are found in the liver. Three weeks after infection, a visible hydatid cyst develops that then slowly grows in a spherical manner. The right lobe is most frequently involved, probably because most of the portal blood from the upper gastrointestinal tract reaches this area. E. multilocularis infection in humans is more serious than E. granulosus, as the parasite produces a multicystic tumour-like infiltrative growth in the liver and, if untreated, carries a mortality approaching 100 per cent.

## THE HYDATID CYST

This is a spherical-shaped collection of fluid housing innumerable scolices. The surrounding parenchyma is displaced, but not invaded, by this expanding colony of scolices which may grow up to a diameter of 30 cm. Surrounding the cyst is an outer layer of adventitia (pericystic layer), which does not belong to the parasite but represents host parenchyma compressed into a fibrovascular layer.

The true parasitic contribution to the hydatid cyst wall is the laminated membrane (ectocyst). This acelluar membrane is glistening white, has the look and feel of a hard boiled egg, is not more than 2 mm thick, very fragile and it is in close contact with the inner surface of the pericyst. Any separation between pericyst and laminated membrane results in rupture and fragmentation of the latter.

On the inside of the cyst, the laminated membrane is lined by a very thin, almost transparent layer, the germinal membrane (endocyst). This cellular layer is the breeding ground for the multiplying scolices. Pedunculated nodes of multiplying cells project into the lumen of the cyst as brood capsules. Scolices develop from these brood capsules and are liberated into the fluid once the connection with the germinal epithelium disrupts. The free floating and gravitating scolices within the cyst fluid are described as hydatid sand. Continuous restructuring of the germinal layer by means of protrusion, invagination, and fragmentation leads to endogenous daughter cysts, and the

original univesicular cyst is eventually filled by hundreds of daughter cysts of various sizes.

The germinal membrane secretes the hydatid fluid, which is a crystal clear transudate of serum that acts as the amniotic fluid for the countless scolices floating within it. It is isotonic, contains protein, and is antigenic. If released into the circulation, eosinophilia, urticaria, bronchospasm, or anaphylaxis may occur. A hydrostatic pressure of 300 to 900 mmH2O can build up within the interior of the cysts, enough to cause a near explosive discharge of the hydatid fluid at operation if the cyst suddenly ruptures. After several years of slowly expanding growth, the hydatid cyst does not enlarge any further and the pericystic adventitial wall may calcify. This would also lead to nutritional deprivation and therefore intracystic death of the larvae.

As hydatid cysts grow by expansion, they tend to make their way towards regions of less resistance. In the liver they protrude from the depth of the organ towards the peritoneal surface, particularly beneath the diaphragm. Pressure necrosis of parenchyma and the wall of tubular structures may eventually result in free rupture into a coelomic cavity (peritoneal or pleural) or into bile ducts, bronchial tree, urogenital system, or vessels.

Although the liver and lungs are most commonly affected, hydatid cysts may occur virtualy anywhere in the body, and may be found in the peritoneal cavity, heart, brain, skeleton, retroperitoneum, pelvic organs, muscles, and surgical incisions.

# CLINICAL FEATURES OF HYDATID DISEASE LIVER

Hydatid cysts are diagnosed in equal numbers of men and women at an average age of about 45.In this series male to female ratio was 1:4. About three fourths of hydatid cysts are located in the right liver and are singular. In this study all the 5 cases have visceral surface of the right lobe of liver involvement. The clinical presentation of a hydatid cyst is largely asymptomatic until complications occur. The symptoms of Hydatid cyst disease are related primarily to the mass effect of the slowly enlarging cyst: abdominal pain from the stretching hepatic capsule, jaundice from compression of the bile duct, or portal hypertension from portal vein obstruction. The most common presenting symptoms are abdominal pain, dyspepsia, and vomiting. The most frequent sign is hepatomegaly (Kalinova.K 2007). In this study 3 cases presented with upper abdominal pain and hepatomegaly was present in all the five cases (One with massive hepatomegaly, Figure 13). Infection of a hydatid cyst can occur and present like a pyogenic abscess. Rupture of the cyst into the biliary tree or bronchial tree or free rupture into the peritoneal, pleural, or pericardial cavities can occur. Free ruptures can result in disseminated echinococcosis and/or a potentially fatal anaphylactic reaction [56,58,59,60].

# INVESTIGATIONS OF HYDATID DISEASE LIVER

- **Eosinophilia** is present in 25 to 35 per cent of patients.
- **The Casoni skin test** was used extensively some years ago is now abandoned.
- **Serological tests** are useful not only for diagnosis and follow-up of hydatid disease but may even be used for screening in high risk populations in endemic areas. The complement fixation test is useful, because it becomes negative after cure of a hydatid cyst and can therefore be used to assess the efficacy of treatment. Enzyme-linked immunosorbent assay (ELISA), indirect haemagglutination, the radioallergosorbent test, and immunoelectrophoresis are also available. These tests was not done in this study.
- **X-ray chest** In endemic areas elevation of right hemi diaphragm in a healthy asymptomatic patient is highly indicative of liver hydatidosis. Calcification of the cyst in the liver is more diagnostic.
- **Ultrasound** is most commonly used worldwide for the diagnosis of echinococcosis. A simple hydatid cyst is well circumscribed with budding signs on the cyst membrane and may contain free floating hyperechogenic hydatid sand. A rosette appearance is seen when daughter cysts are present. Calcifications in the wall of the cyst are highly suggestive of hydatid disease and can be helpful in the diagnosis (Figure 19). USG was done in all the cases [29,30].

## Classification of Ultrasound appearances in Hydatid disease (GHARBI)[30]

| Type | Description |
|---|---|
| I | Pure fluid collection (non-complicated unilocular cyst) |
| II | Fluid collection with a split wall (Detached membrane) |
| III | Fluid collection with daughter cysts (honeycomb image) |
| IV | Heterogeneous solid echo pattern with thick membranes and few daughter cysts |
| V | Calcified non-viable degenerated cyst. |

- **CT or MRI** CT scans shows the Hydatid cyst as a sharply defined, low-density lesion with spoke-like septations. The presence of a calcified rim of daughter cysts greatly enhances the specificity (Figure 20). These studies can also evaluate extra hepatic disease and demonstrate detailed hepatic anatomic relationships to the cyst. The real value of MRI is in skeletal and vertebral hydatidosis. CT scan was done in all the cases.

- **ERCP or Percutaneous Transhepatic Cholangiography (PTC)** may be necessary in patients with suspected biliary involvement. Persistent postoperative biliary fistulas may be diagnosed and treated by ERCP with endoscopic sphincterotomy.

# TREATMENT OF HYDATID DISEASE LIVER

## CHEMOTHERAPY

Chemotherapy for echinococcosis with albendazole, mebendazole (400 to 600 mg daily for 3 to 4 weeks) or Praziquantel (60 mg/kg for up to 2 weeks) is effective in 20% to 30% of patients infected with E. granulosus but requires long-term treatment. Albendazole is given as 4-week cycles, separated by 2-week drug-free intervals, in a dose of 10 to 15 mg/kg per day, the drug is continued for several cycles until sterilization or regression of the hydatid process is documented by ultrasonography or radiology. Chemotherapy should generally be considered for widely disseminated disease or patients with poor surgical risk [35].

**PAIR** (**P**ercutaneous **A**spiration, **I**nfusion of scolicidal agents, and **R**easpiration)

In recent years, a number of authors have reported percutaneous aspiration and injection of scolicidal agents with early success rates about 70%. PAIR is now recommended instead of surgery. PAIR is contraindicated for superficially located cysts (because of the risk of rupture), for cysts with multiple thick internal septal divisions (honeycombing pattern), and for cysts communicating with the biliary tree. For prophylaxis of secondary peritoneal echinococcosis due to inadvertent spillage of fluid during PAIR, the administration of albendazole (15 mg/kg daily in two divided doses) should be

initiated at least 4 days before the procedure and continued for at least 4 weeks afterward. This procedure was not practiced in our institution [33, 34].

**SURGERY -** Surgery remains the gold standard treatment for hydatid liver disease. The aim of surgical intervention is to inactivate the parasite, to evacuate the cyst along with resection of the germinal layer, to prevent peritoneal spillage of scolices and to obliterate the residual cavity [64,65,66,67].

**Indications for operation:**

- Patient condition
- Cyst characteristic

The natural history of viable hydatid cysts is one of the growth and potential complication, a middle aged; asymptomatic, healthy patient is an operative candidate. An elderly patient with small, asymptomatic, calcified cysts, conservative management is appropriate.

**The surgical treatments employed** are

- Partial cystectomy & tube drainage
- Partial cystectomy with capitonnage
- Partial cystectomy with capitonnage and omentoplasty
- Cystopericystectomy
- Marsupialization (Lindemann's Procedure)
- Liver resection

In this study, Partial cystectomy with capitonnage & Marsupialization was done for two cases each & Partial cystectomy with tube drainage was done for one case.

Prior to surgery Albendazole 400 mg OD was given for 4 days and was continued for 6 weeks postoperatively. The anesthesiologist should have epinephrine and corticosteroids available for the potential of an anaphylactic reaction. A number of operations have been utilized, but, in general, the abdomen is completely explored by Right Subcostal or upper midline incision, the liver mobilized, and the cyst exposed. Packing of the abdomen with pads soaked in Scolicidal agents is done, because rupture can result in anaphylaxis and diffuse seeding. The cyst was then aspirated through a closed suction system and flushed with a scolicidal agent such as hypertonic saline (3, 5, 10, 20, or 30 per cent), silver nitrate (0.5 per cent), hydrogen peroxide (10 per cent), povidone iodine (10 per cent), diluted Savlon (0.5 per cent cetrimide with 0.05 per cent chlorhexidine), praziquantel, absolute alcohol (96 per cent), and others. Whether any of these is really 100 per cent scolicidal remains unclear. In this study 3% hypertonic saline was used as scolicidal agent. After some 5 to 10 min contact time, the scolicidal fluid is aspirated and the cyst cavity is evacuated meticulously using spoon, leaving the pericyst lining the cavity. After evacuation the cyst cavity is washed again with 3% hypertonic saline and looked for any bile leak. The cyst is then unroofed, which can then be followed by a number of possibilities, depending on its size and site. Simple cyst closure & tube drainage, omentoplasty, capitonnage, marsupialization procedures, Roux-en-Y Cystojejunostomy or even liver resection.

In **OMENTOPLASTY**, omentum is mobilised from the transverse colon, providing sufficient length to pack the cavity. The omentum is sutured into place; a drain is passed together with the pedicle in to the cavity (Seyed Reza Mousavi et al, 2005). The drain was then fixed to the rim of the opening so that its tip is in the most dependent portion of the cavity. In this study this procedure has not been performed.

If the omentum is not available, the cyst cavity can be obliterated by **CAPITONNAGE**. This technique involves in folding redundant cyst wall in the depths of the cyst by successive layers of sutures. This is not possible if the cyst walls are calcified and rigid. In this study, this procedure was performed for 2 cases and drain was removed on the $5^{th}$ Post Operative Day.

**MARSUPIALIZATION** consists of suturing the adventitia to the parietes, thereby allowing free and direct drainage of the cavity to the exterior. This procedure is ideal for infected cysts. The procedure is safe, but convalescence is slow and drainage may persist for months. This procedure was performed for two cases (Figure 21 to 24) and postoperative bile leak was present, but spontaneous cessation occurs on $5^{th}$ to $6^{th}$ postoperative day.

**CYSTOPERICYSTECTOMY** is ‘’en block’’ excision of the hydatid cyst including the adventitia. The plane between the adventitia and the liver parenchyma can be developed easily but bleeds profusely. The recurrence and morbidity rates are low and it can be done with an acceptable operative

mortality in experienced hands. This procedure is to be avoided for cysts impinging on the hepatic veins, IVC and liver hilum. In this study this procedure has not been performed.

**HEPATIC RESECTION** is reserved for recurrent disease. Resection or liver transplantation is also the best choice for the treatment of E. multilocularis because of the lack of demarcation in the liver and its aggressive infiltrative growth.

When bile duct communication is diagnosed at operation or preoperatively, it must be meticulously searched for. Simple suture repair is often sufficient, but major biliary repairs or approaches through the common bile duct may be necessary. Internal drainage with a Roux-en-Y jejunal loop may be used for deep-seated hepatic cysts with biliary connections.

Laparoscopic techniques for drainage and unroofing of cysts have been reported in a number of series with encouraging results. Laparoscopic evacuation of the hydatid cysts can be achieved by a special aspiration–grinder apparatus.

## Complications of Surgery

Wound infection and abscess formation in infected cases. Postoperative biliary fistula in some cases, a small amount of biliary drainage is common and should not cause concern if volume is low. Drains are left in place and spontaneous cessation is a rule (M.I.Yildirgan et al, 2003). In this study among 5 cases, 2 cases developed postoperative low volume biliary leak from the drain tube, which was managed conservatively and in both cases spontaneous cessation occurs on the $5^{th}$ to $6^{th}$ POD.

**Recurrence** rates after surgical treatment range from 1% to 20% but are generally 5% or less in experienced centers. Recurrence could not be assessed because our patients are irregular in follow up.

## Prevention of Echinococcosis

In endemic areas, echinococcosis can be prevented by administering praziquantel to infected dogs, by denying dogs access to infected animals, or by vaccinating sheep. Limitation of the number of stray dogs is helpful in reducing the prevalence of infection among humans.

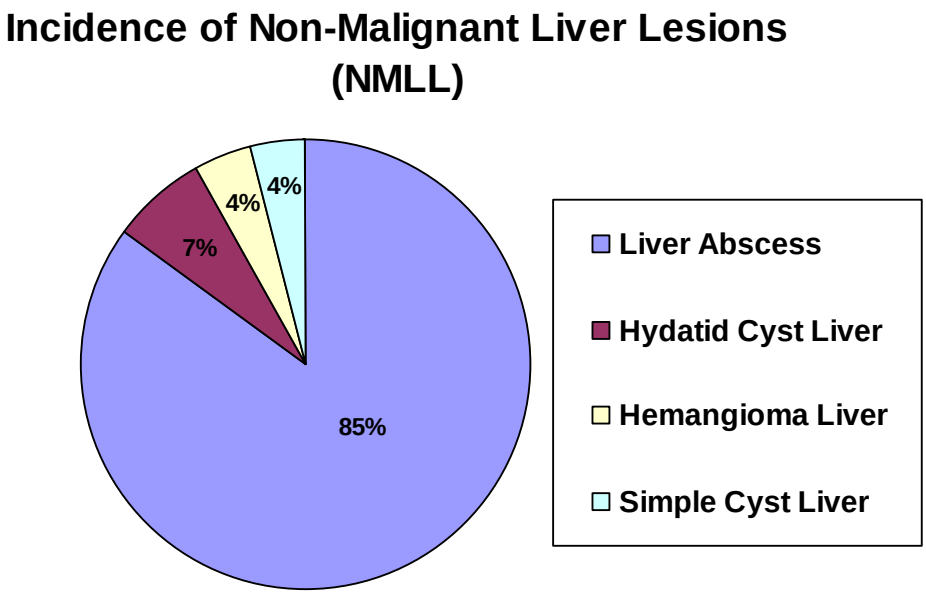
# MATERIALS AND METHODS

A study of 75 cases of Non malignant liver lesions was undertaken among the patients admitted in Surgical wards of Coimbatore Medical College Hospital during the period of July 2006 to august 2008. Since the non infective part of non malignant liver lesions were of very low incidence and all those lesions were detected incidentally on radiological evaluation of the patient for some other diseases and were managed conservatively in our institution this part was omitted in this study. Studies in the incidence, clinical presentation, investigations, complications and treatment of Infective Non malignant lesions such as Liver abscess and Hydatid disease were undertaken. The study includes patients with age group above 12 years and both sex. The cases were studied in detail using a proforma under the guidance of my unit chief.

Diagnosis of Infective Non malignant lesions was based on history, physical examination, ultrasonographic evaluation and in some cases computerised axial tomography (C.T.) scan and on aspiration of the abscess from the liver which confirms the radiological and clinical diagnosis. Using a standard pro forma, patients presenting symptoms and signs, haematological and biochemical results, microbiological findings, diagnostic method findings, abscess characteristics, treatment were documented.

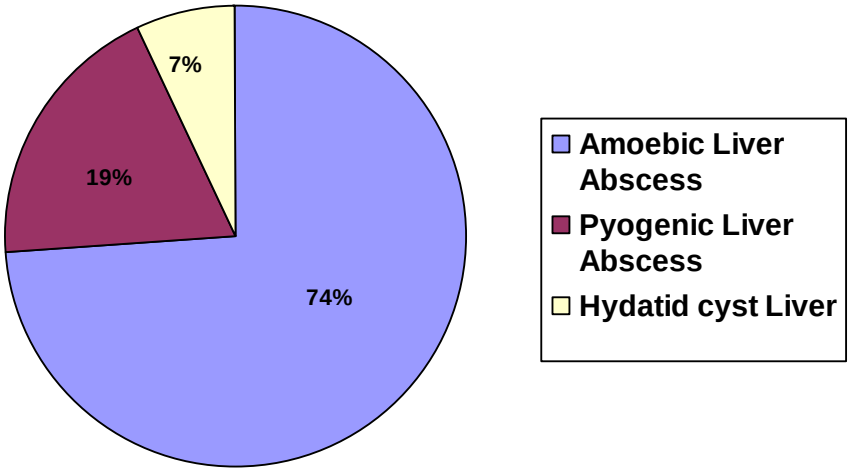
# OBSERVATIONS AND DISCUSSION

In this study of 75 cases of non-malignant liver lesions, only six cases (8%) were of non-infective component and remaining 69 cases (92%) were infective one.

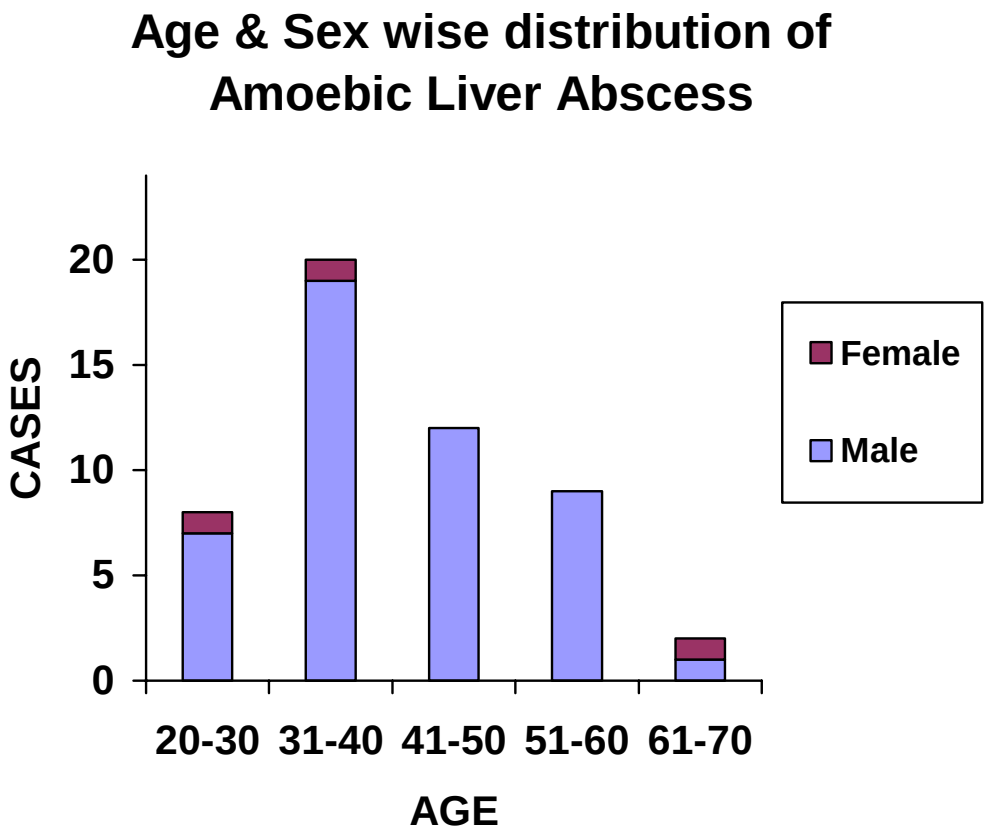



| NMLL | PERCENTAGE | CASES |
|---|---|---|
| Liver Abscess | 85% | 64 |
| Hydatid Cyst Liver | 7% | 5 |
| Hemangioma Liver | 4% | 3 |
| Simple Cyst Liver | 4% | 3 |
| Total Cases | | 75 |

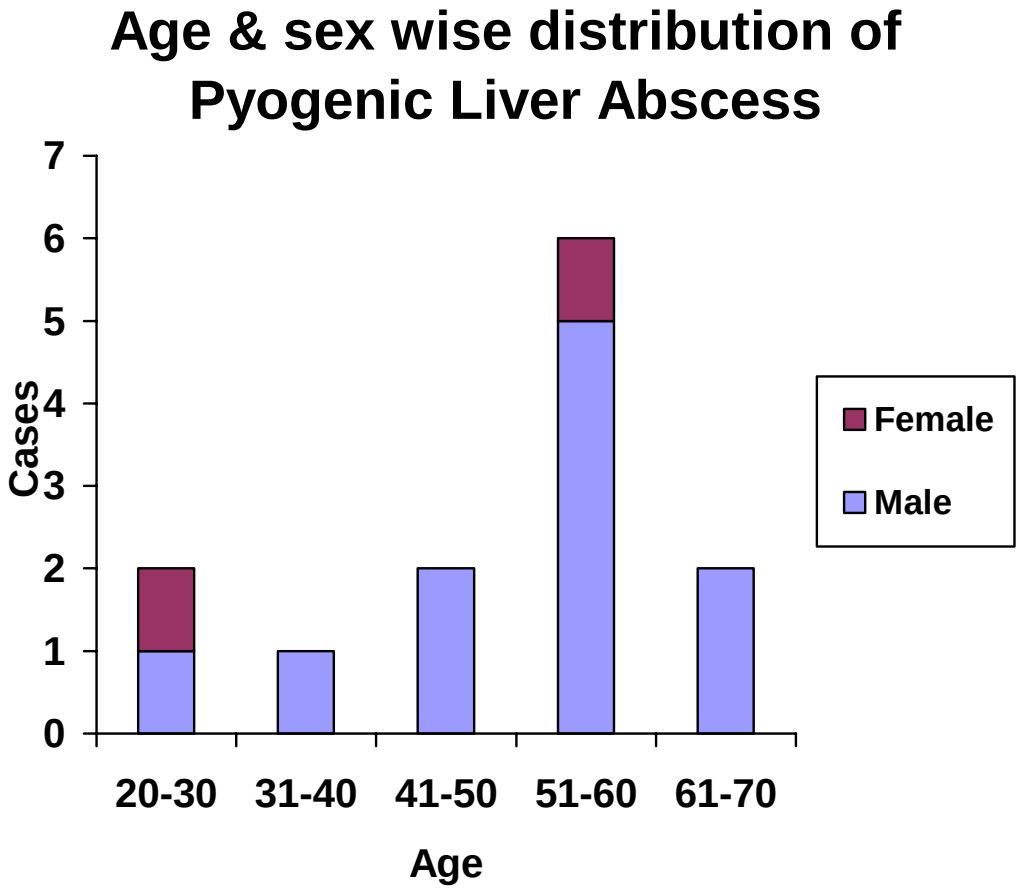
## Incidence Infective Non-Malignant Liver Lesions




| INFECTIVE NMLL | PERCENTAGE | CASES |
|---|---|---|
| Amoebic Liver abscess | 74% | 51 |
| Pyogenic Liver Abscess | 19% | 13 |
| Hydatid Cyst Liver | 7% | 5 |
| Total Infective NMLL cases | | 69 |




In this study peak incidence of Amoebic liver abscess was found between 3rd & 4th decade of life and about 94% (48 cases) of patients were male.




In this study peak incidence of Pyogenic Liver Abscess was found between 5th & 6th decade of life and about 85%(11 cases) cases were male.

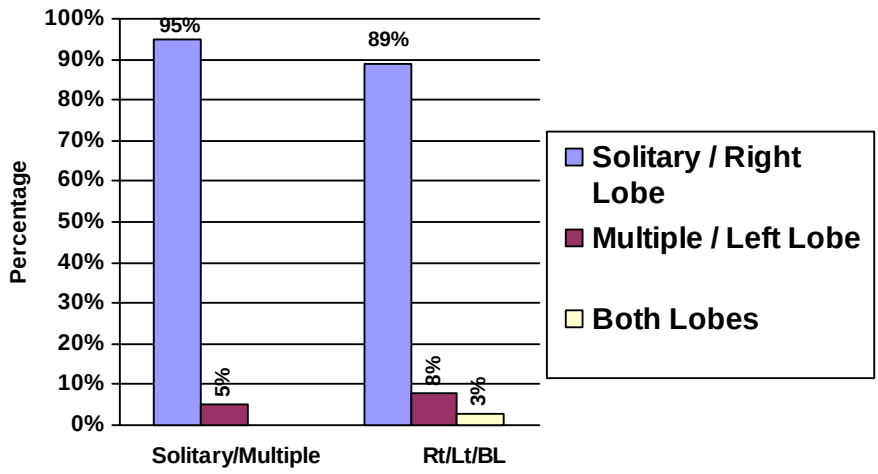
## CLINICAL PRESENTATION OF LIVER ABSCESS

| Clinical Presentation | Cases | Percentage (%) |
|---|---|---|
| **Fever / Chills** | 54 | 85 |
| Nausea / Vomiting | 29 | 45 |
| **Abdominal Pain** | 58 | 90 |
| Anorexia / Weight Loss | 19 | 30 |
| Diarrhea | 9 | 15 |
| Cough | 3 | 5 |
| **Intercostals tenderness** | 45 | 70 |
| **Hepatomegaly** | 45 | 70 |
| Peritoneal Signs | 2 | 4 |
| Jaundice | 6 | 10 |

## COMBINATION OF CLINICAL PRESENTATION

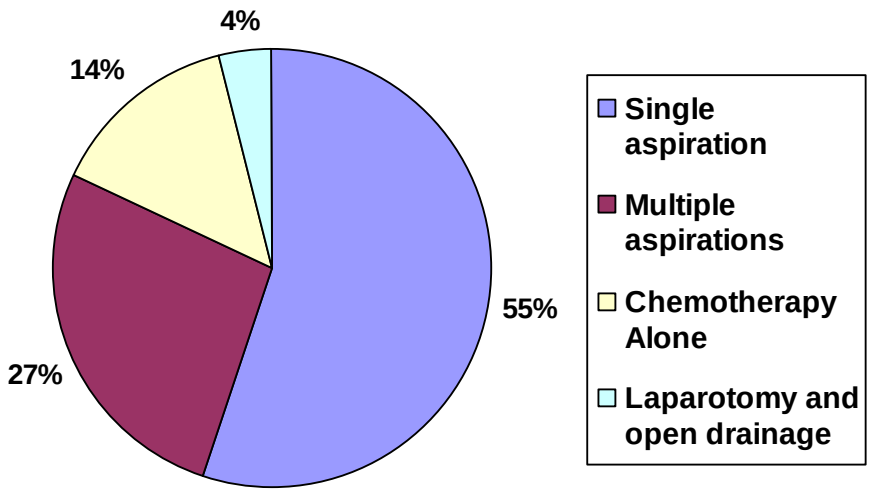
| | |
|---|---|
| Total no. of cases | 64 |
| Fever & Abdominal Pain | 48 |
| Intercostals tenderness & Hepatomegaly | 35 |

## Solitary vs Multiple Abscess
## Right vs Left lobe abscess




| SOLITARY/MULTIPLE | PERCENTAGE | CASES |
|---|---|---|
| Solitary liver abscess | 95% | 61 |
| Multiple liver abscess | 5% | 3 |
| Total Cases | | 64 |

| RT /LT / BOTH LOBES | PERCENTAGE | CASES |
|---|---|---|
| Right lobe liver abscess | 89% | 57 |
| Left lobe liver abscess | 8% | 5 |
| Both lobe abscess | 3% | 2 |
| Total Cases | | 64 |




Single aspiration (35 cases) (55%)

Multiple aspirations (17 cases) (27%)

Chemotherapy alone (9 cases) (14%) was given for abscess < 5cm and all the cases responded well.

Laparotomy (2 cases) (3%)

ICD insertion and Laparotomy (1 case) (1%)

In this study, for liver abscesses <5cm chemotherapy alone (9 cases) was given, for liver abscesses >5cm and left lobe abscess, Ultrasound guided percutaneous needle aspiration (52 cases) was done and for complicated cases (3 cases) laparotomy was done.

# METHODS OF MANAGEMENT OF LIVER ABSCESS

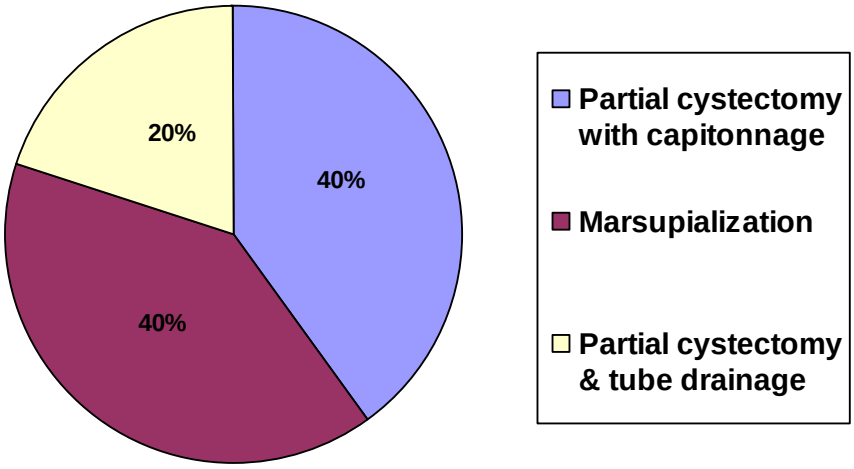
| MANAGEMENT | CASES | PERCENTAGE (%) |
|---|---|---|
| Single aspiration | 35 | 55% |
| Multiple aspiration | 17 | 27% |
| Laparotomy and open drainage | 2 | 3% |
| Inter costal tube drainage and Laparotomy | 1 | 1% |
| Chemotherapy alone | 9 | 14% |

All these patients were supplemented with Intra venous antibiotics (Combinations of a Cephalosporin, an aminoglycoside, and metronidazole).
All cases were aspirated in the operation theatre after marking the site of aspiration with ultrasound.

Three cases presented with complication, two with intra-peritoneal rupture and one with rupture in to the right side pleural cavity. Peritoneal rupture was dealt with an emergency laparotomy and thorough peritoneal lavage was given after placing a Mallecot's catheter in the abscess cavity. These patients had uneventful postoperative period and drain was removed on the 7$^{th}$ day.

In one case with rupture in to the pleural cavity, ICD was inserted, but on the 3$^{rd}$ day after the ICD insertion, the patient developed peritonitis so laparotomy was planned. On laparotomy there was purulent exudate from rupture of right sided liver abscess, which was thoroughly aspirated and peritoneal lavage was given. Mallecot's catheter was placed in the abscess cavity. This patient had a very stormy postoperative period but he recovered well later and was discharged.

## Management of Hydatid cyst Liver




Total cases – 5

Partial cystectomy & tube drainage - 1

Partial cystectomy with capitonnage - 2

Marsupialization – 2

In this study among five cases, two cases for which Marsupialization have been done developed postoperative low volume biliary leak from the drain tube, which was managed conservatively and in both cases, spontaneous cessation occurs on the 5$^{th}$ to 6$^{th}$ postoperative period.

# CONCLUSION

- Amoebic liver abscess was predominant in adult males in the 3rd & 4th decades of life.
- Pyogenic liver abscess was equally prevalent in both sexes in the 5th &6th decades of life.
- Most cases of liver abscess presented with fever and abdominal pain and on examination intercostal tenderness and hepatomegaly was elicited.
- Ruptured liver abscess presented as acute abdomen in two cases.
- Amoebic liver abscess had a highly variable Clinical presentation. Delayed diagnosis resulted in rupture of abscesses. High index of clinical suspicion combined with Ultrasound of abdomen was helpful in reaching an early diagnosis.
- Chemotherapy and percutaneous needle aspiration were sufficient for most uncomplicated amoebic and pyogenic liver abscess in this study.

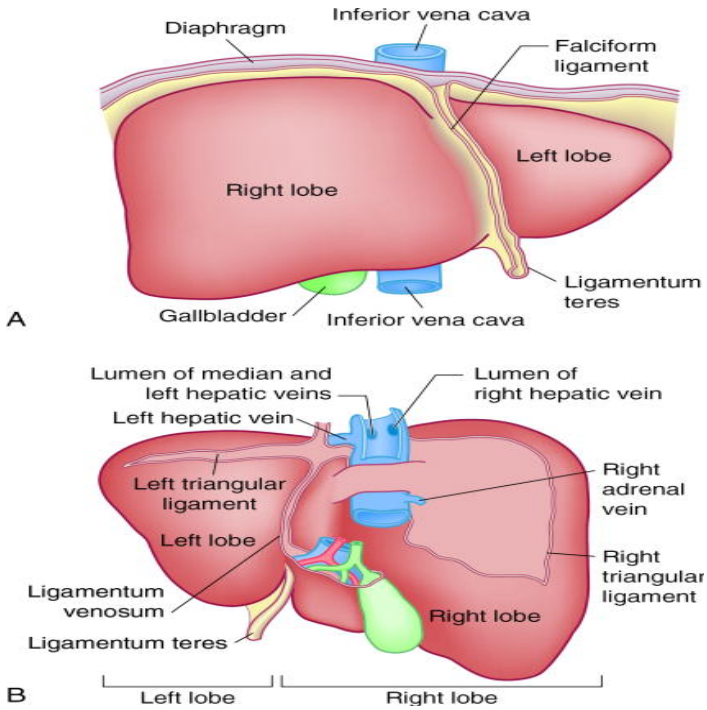



**Figure-1 A,** Historically, the liver was divided into right and left lobes by the external marking of the falciform ligament. **B,** The posterior and inferior surface of the liver is shown.

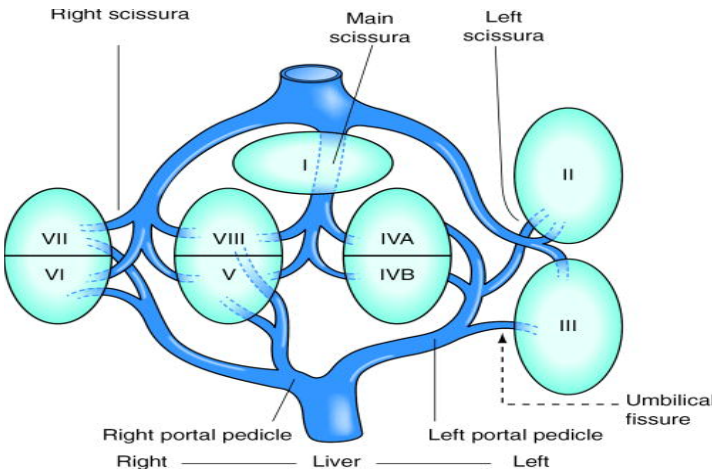



**Figure 2** Schematic diagram of the segmental anatomy of the liver. The eight segments are illustrated and the four sectors, divided by the three main hepatic veins running in scissurae, are shown. The umbilical fissure (not a scissura) is shown to contain the left portal pedicle.

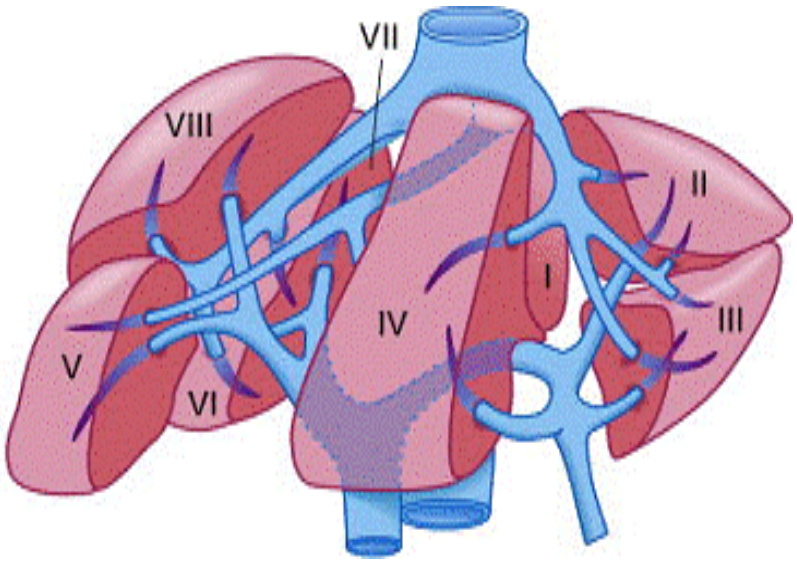



**Figure 3** Segmental anatomy of the liver, as seen at laparotomy in the anatomic position.

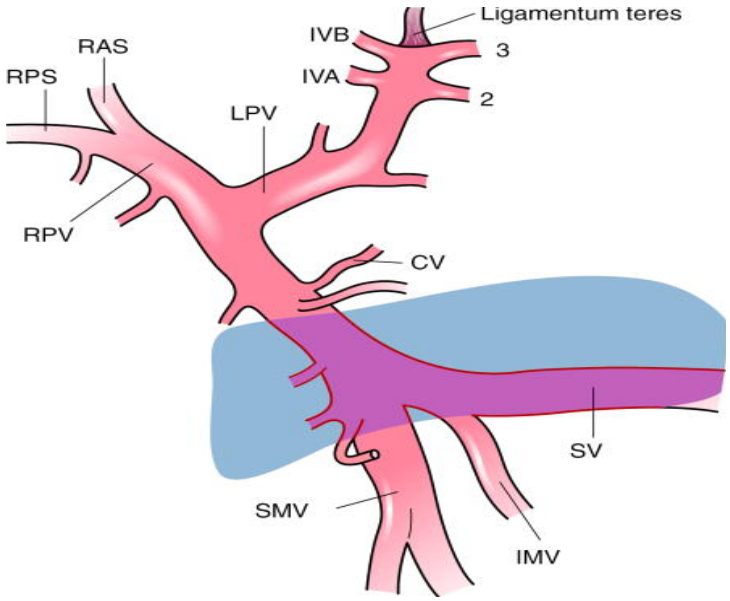



**Figure 4** The anatomy of the portal vein is demonstrated. The superior mesenteric vein (SMV) joins the splenic vein (SV) posterior to the neck of the pancreas *(shaded)* to form the portal vein.

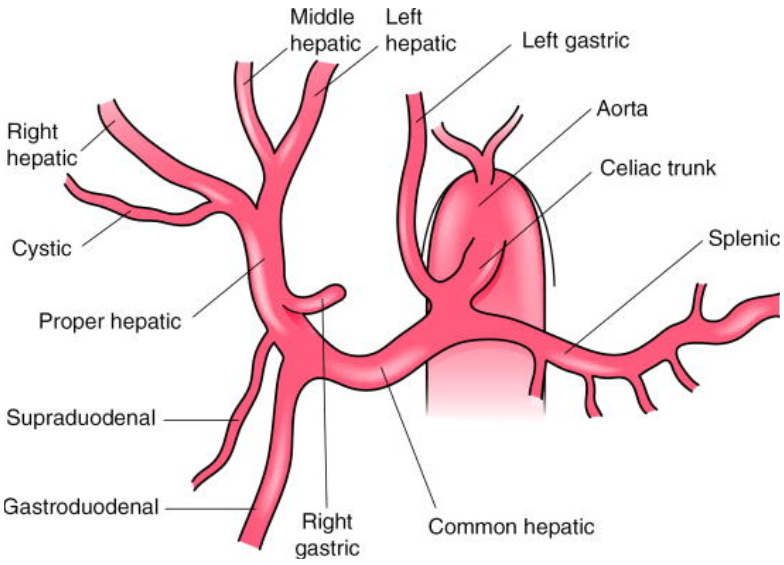



**Figure 5** The most common anatomy of the celiac axis and hepatic arterial system is demonstrated. The celiac axis, just below the diaphragmatic hiatus, trifurcates into the splenic, left gastric, and common hepatic arteries.

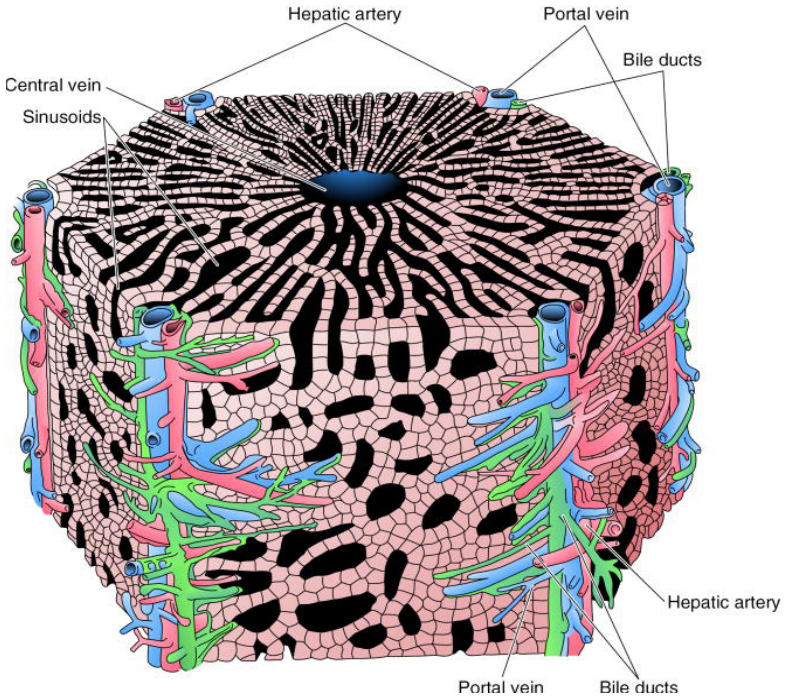



**Figure 6** Hepatic Lobule

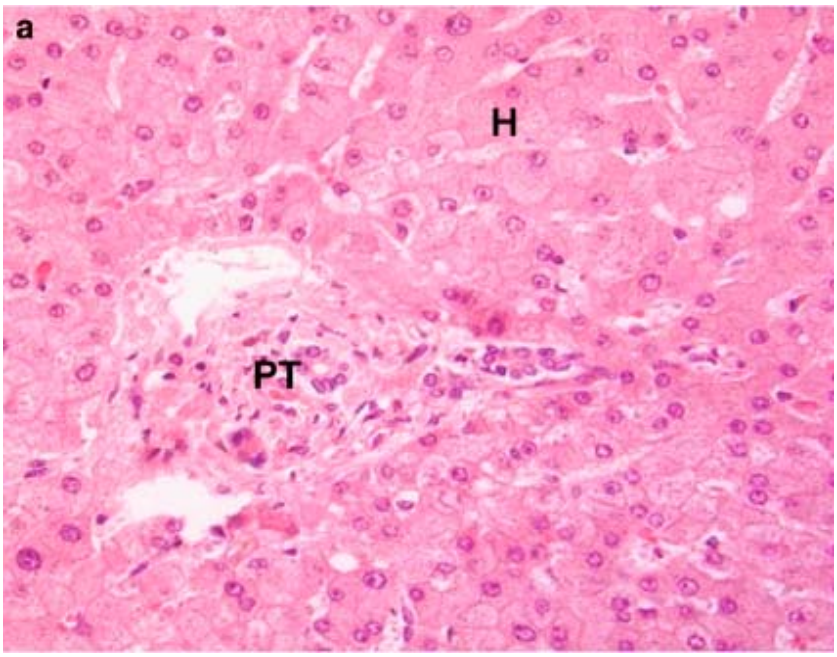



**Figure 7** Histology of Liver, H – Hepatocytes, PT – Portal Triad

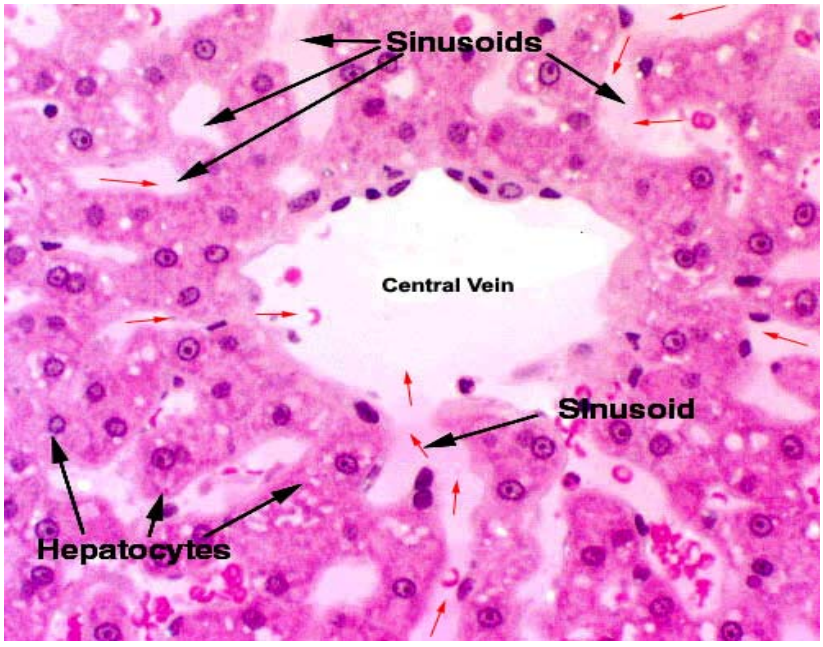



**Figure 8** Hepatic Sinusoids

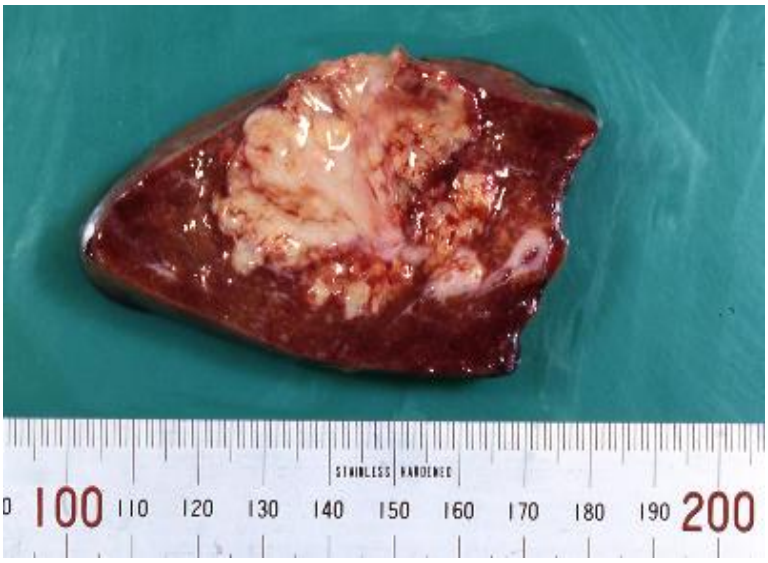



**Figure 9** Specimen of Rt lobe Amoebic liver abscess

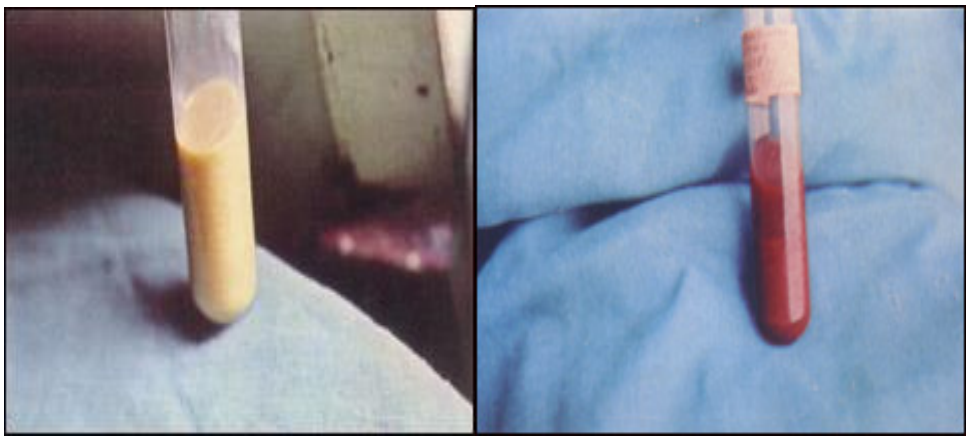


**Figure 10** Thick yellow pus of Pyogenic liver abscess and Anchovy sauce pus of Amoebic liver abscess.

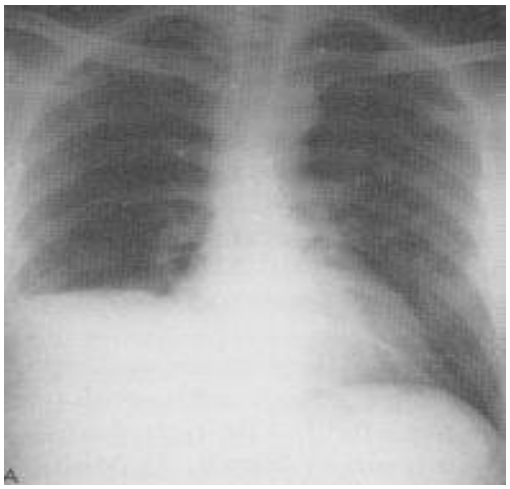


**Figure 11** Plain Chest radiograph demonstrates an elevated right hemi diaphragm & Rt pleural effusion in a patient with an amoebic liver abscess.

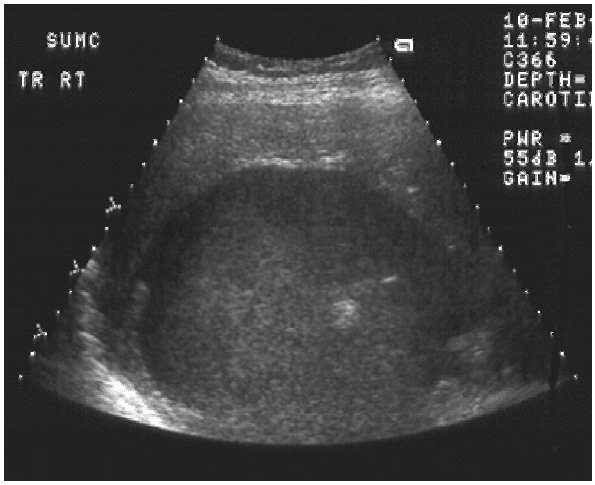



**Figure 12** Ultrasound-a rounded hypoechoic and nonhomogeneous lesion (Amoebic liver abscess)

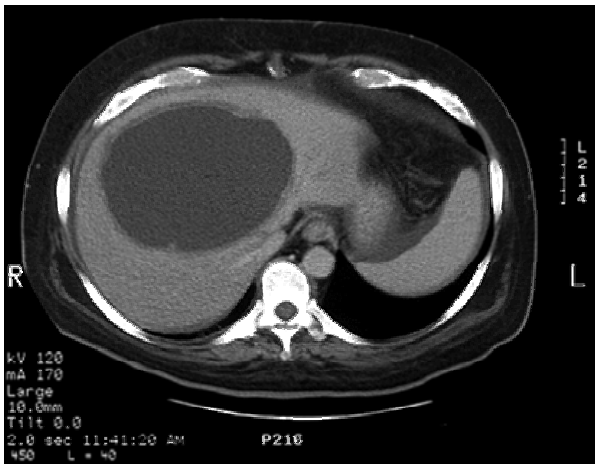



**Figure 13 A** Plain CT scan demonstrates an amoebic abscess in the right liver.

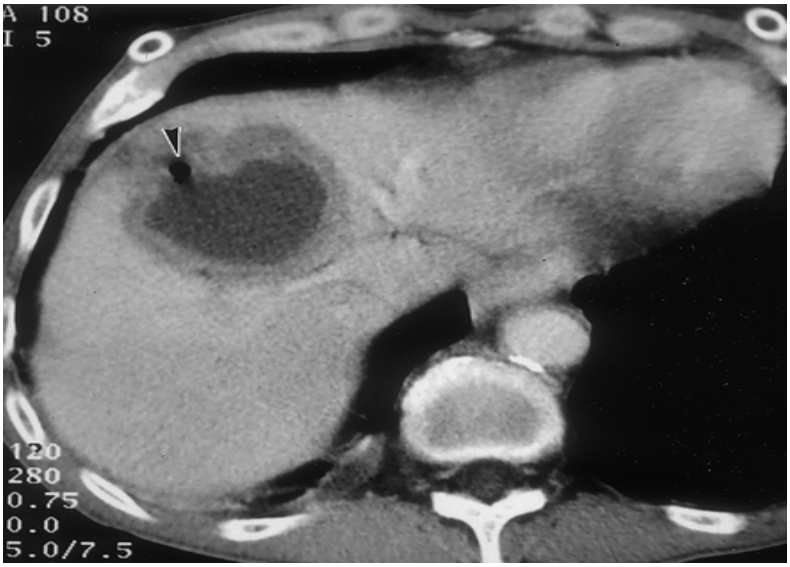



**Figure 13 B** Contrast-enhanced CT scan shows a thick-walled **cystic** lesion with peripheral enhancement. Note the small pocket **of** air in the nondependent portion **of** the mass (arrowhead), which is virtually a pyogenic liver abscess.

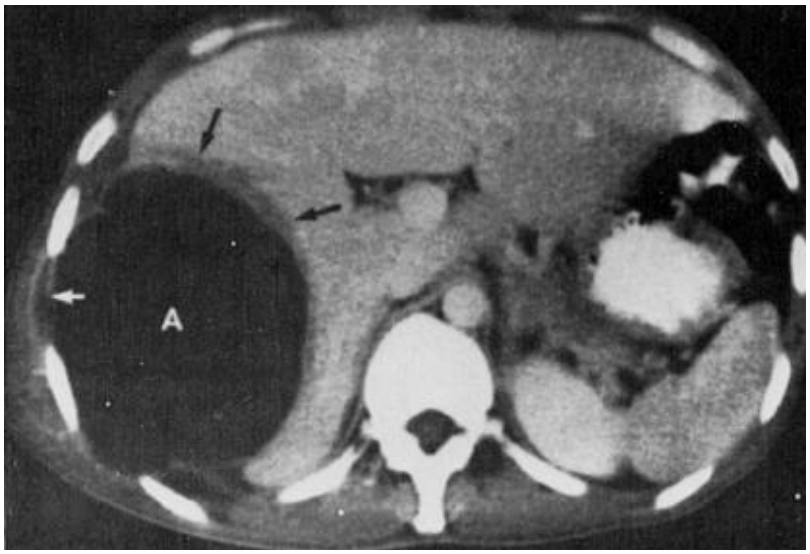



**Figure 14** Contrast-enhanced CT scan of amebic abscess (A). The lesion is peripherally located and round. Rim is nonenhancing but shows peripheral edema (black arrows). Note the extension into the intercostal space (white arrows).

**Figure 15** A case of Liver Abscess

**Figure 16** Ultrasound marking of the site of aspiration

**Figure 17** Percutaneous aspiration of a Pyogenic liver abscess case

**Figure 18** Pyogenic Liver Abscess

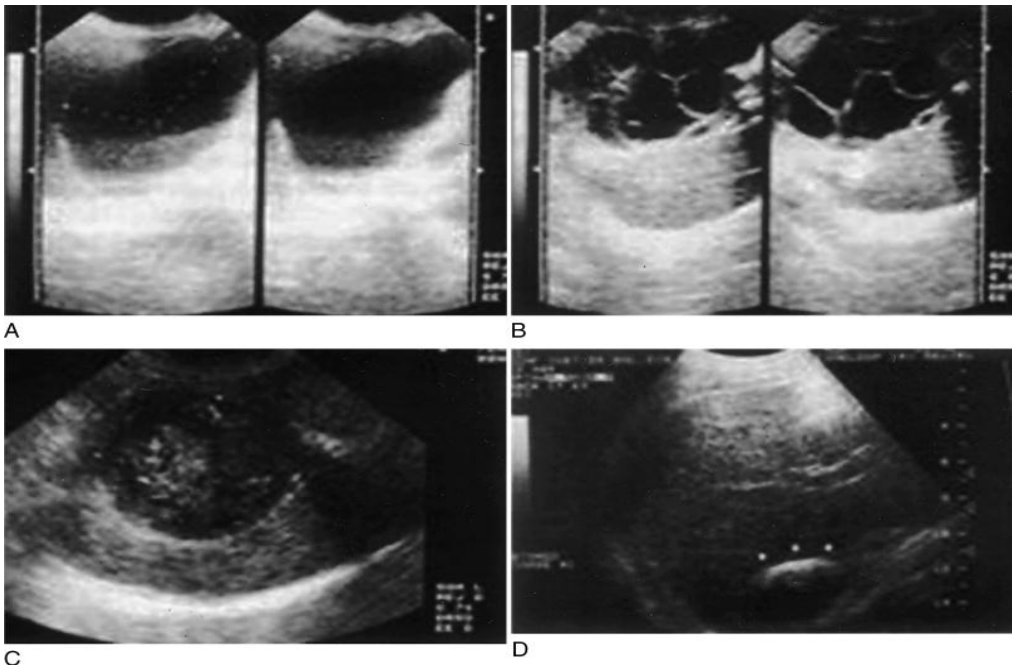



**Figure 19** Ultrasound demonstrating typical characteristics of hydatid cyst at varying stages. A, Simple hydatid cyst with “hydatid sand.” B, Daughter and granddaughter cysts and typical rosette appearance. C, Hydatid cyst filled with amorphous mass giving a solid or semi-solid appearance. D, Calcified cyst with “eggshell” appearance.

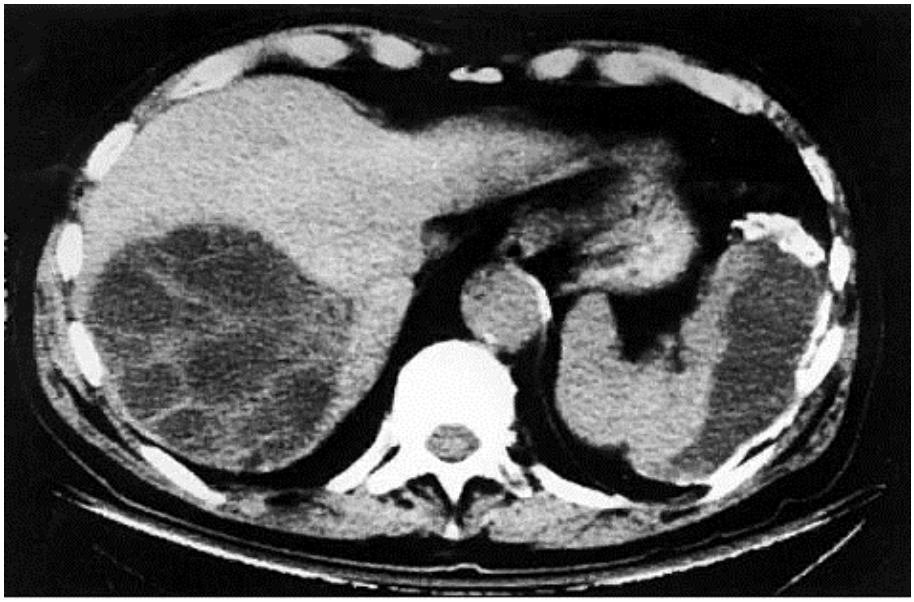


**Figure 20** Abdominal CT scan showing a large primary Hydatid cyst (Echinococcus granulosus) in the right lobe of the liver. Note multiple internal septations indicating secondary (daughter) cyst formation.

**Figure 21** A case of Hydatid disease Liver with Massive Hepatomegaly

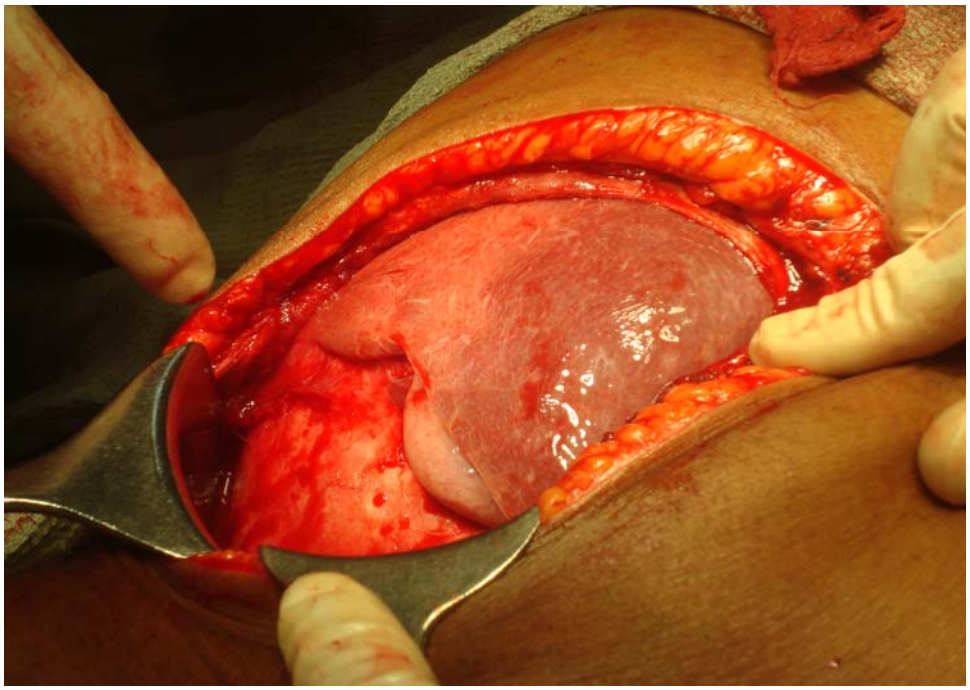


**Figure 22** A huge Hydatid cyst on visceral surface of the liver

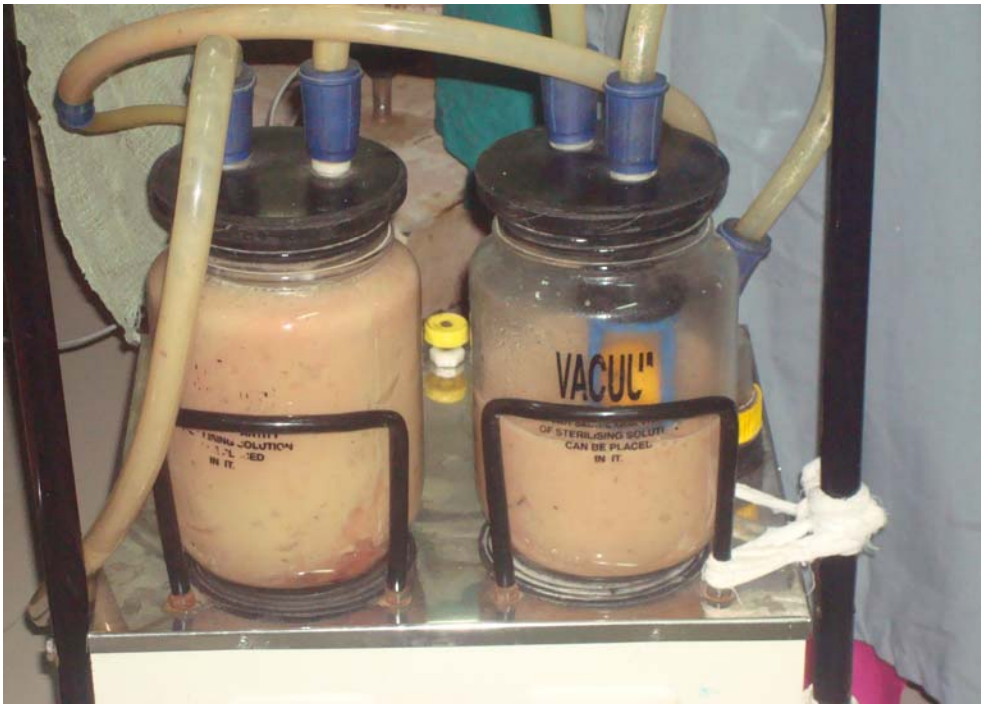


**Figure 23** Aspirated Hydatid sand in the suction apparatus

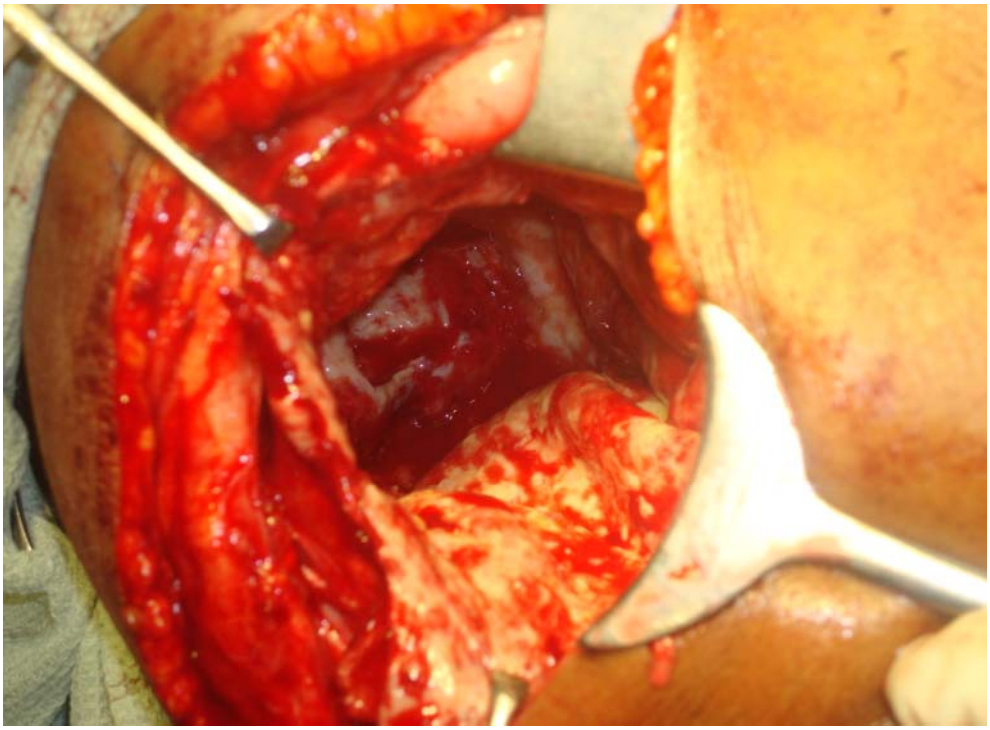


**Figure 24** Evacuated Cyst Cavity

**Figure 25** Evacuated Brood Capsules

**Patient S.No.** 69 45Yrs/ M **IP No.** 43957 **Operated On:** 16.07.2008

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# PROFORMA

NAME: AGE/SEX:

IP NO: WARD

**PRESENTING FEATURES:**

FEVER
CHILLS
ANOREXIA
JAUNDICE
RIGHT UPPER QUADRENT ABDOMINAL PAIN
NAUSEA
VOMITING
DIARRHEA
WEIGHT LOSS
SHOULDER PAIN
COUGH

H/O PREVIOUS TREATMENT (SURGERY, DRUGS, Etc.)

**GENERAL EXAMINATION:**

ANAEMIA
ICTERUS
FEBRILE
BP
PR
RR

**EXAMINATION OF ABDOMEN:**

RIGHT UPPER QUADRENT TENDERNESS
HEPATOMEGALY
INTERCOSTALS TENDERNESS
PERITONEAL SIGNS

**EXAMINATION OF OTHER SYSTEMS:**

RS: RALES, DECREASED BREATH SOUNDS,
DULLNESS TO PERCUSSION

CVS

CNS

**INVESTIGATIONS:**

COMPLETE HEMOGRAM

MOTION EXAMINATION

LIVER FUNCTION TEST

CULTURE OF ABSCESS

X-RAY CHEST

X-RAY ABDOMEN

ULTRASONOGRAM ABDOMEN

CT SCAN ABDOMEN

SEROLOGY

DIAGNOSTIC ASPIRATION

**TREATMENT:**

**MEDICAL**

**ULTRA SOUND GUIDED PERCUTANEOUS DRAINAGE**

**SURGICAL**

# MASTER CHART

| S.No | Name | Age | Sex | IP No. | Diagnosis | Treatment |
|---|---|---|---|---|---|---|
| 1 | Velligiri | 60 | M | 57496 | ALA | PNA |
| 2 | Inidharaj | 35 | M | 58931 | ALA | CT |
| 3 | Ramasamy | 60 | M | 36052 | PLA | PNA |
| 4 | Muthu | 40 | M | 52459 | ALA | PNA |
| 5 | Moorty | 40 | M | 47528 | ALA | CT |
| 6 | Kanappan | 51 | M | 3167 | PLA | PNA |
| 7 | Muthusamy | 45 | M | 33314 | ALA | PNA |
| 8 | Manikam | 60 | M | 44828 | PLA | PNA |
| 9 | Rajan | 33 | M | 47810 | ALA | PNA |
| 10 | Muthamal | 52 | F | 2425 | SCL | Conservative |
| 11 | Ganesan | 45 | M | 3932 | ALA | PNA |
| 12 | Kannan | 43 | M | 3896 | ALA | PNA |
| 13 | Rahavan | 41 | M | 16675 | ALA | CT |
| 14 | Kathirvel | 45 | M | 5468 | Ruptured ALA | EL |
| 15 | Tamilarasan | 35 | M | 46638 | ALA | PNA |
| 16 | Subramani | 47 | M | 43347 | PLA | PNA |
| 17 | George manoharan | 48 | M | 52579 | ALA | PNA |
| 18 | Murugan | 31 | M | 8547 | HL | Conservative |
| 19 | Nataraj | 42 | M | 2560 | ALA | PNA |

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|---|---|---|---|---|---|---|
| 20 | Krisnamoorty | 58 | M | 2581 | ALA | PNA |
| 21 | Arasi | 29 | F | 4901 | HCL | PC&TD |
| 22 | Ramu | 51 | M | 92447 | ALA | PNA |
| 23 | Selvaraj | 45 | M | 55017 | ALA | PNA |
| 24 | Paramasivan | 55 | M | 59025 | PLA | PNA |
| 25 | Muthu | 40 | M | 59914 | ALA | PNA |
| 26 | Rathinam | 38 | M | 2163 | Ruptured ALA | EL |
| 27 | Sukumar | 36 | M | 61328 | ALA | PNA |
| 28 | Arputhasamy | 65 | M | 672089 | PLA | PNA |
| 29 | Leelavathi | 60 | F | 68169 | HCL | PC&C |
| 30 | Palanisamy | 49 | M | 19355 | ALA | PNA |
| 31 | Murugan | 35 | M | 25490 | ALA | CT |
| 32 | Solaiammal | 60 | F | 28121 | ALA | PNA |
| 33 | Chandran | 49 | M | 9496 | SCL | Conservative |
| 34 | Sukumar | 30 | M | 51399 | ALA | PNA |
| 35 | Ayyasamy | 44 | M | 55752 | PLA | PNA |
| 36 | Sugumar | 36 | M | 61328 | ALA | CT |
| 37 | Suresh | 57 | M | 62706 | ALA | PNA |
| 38 | Muthusamy | 65 | M | 71684 | ALA | PNA |
| 39 | Shafi | 33 | M | 65214 | ALA | PNA |
| 40 | Muthulaksmi | 30 | F | 380 | HL | Conservative |
| 41 | Maral | 35 | F | 52716 | HCL | Marsupialization |
| 42 | Suklan | 58 | M | 1731 | ALA | PNA |
| 43 | Nagamani | 51 | M | 68169 | HCL | PC&C |

| | | | | | | |
|---|---|---|---|---|---|---|
| 44 | Nagaraj | 22 | M | 31671 | PLA | PNA |
| 45 | Murugesan | 60 | M | 34957 | ALA | PNA |
| 46 | Valarmathi | 29 | F | 40626 | ALA | CT |
| 47 | Palanisamy | 32 | M | 4782 | ALA | PNA |
| 48 | Mohamed ali | 56 | M | 6123 | PLA | PNA |
| 49 | Ayaasamy | 41 | M | 17779 | ALA | PNA |
| 50 | Maduraiveeran | 45 | M | 19267 | ALA | PNA |
| 51 | Subramani | 27 | M | 4450 | ALA | CT |
| 52 | Xavier | 43 | M | 5879 | ALA | PNA |
| 53 | Manivel | 40 | M | 28813 | ALA with rupture into P&A C. | ICD&EL |
| 54 | Rayappan | 40 | M | 42624 | ALA | PNA |
| 55 | Palani | 45 | M | 45659 | ALA | PNA |
| 56 | Vasuki | 35 | F | 35174 | ALA | PNA |
| 57 | Sankili | 52 | F | 35196 | PLA | PNA |
| 58 | Subramani | 55 | M | 2470 | SCL | Conservative |
| 59 | Kerthana | 21 | F | 36991 | PLA | PNA |
| 60 | Ramu | 35 | M | 18531 | ALA | PNA |
| 61 | Natchimuthu | 70 | M | 29743 | PLA | PNA |
| 62 | Mohamed ali | 51 | M | 32499 | ALA | PNA |
| 63 | Natchimuthu | 26 | M | 45036 | ALA | PNA |
| 64 | Krishana menon | 63 | M | 46660 | ALA | PNA |
| 65 | Ayyasamy | 38 | M | 50399 | ALA | CT |
| 66 | Nagaraj | 53 | M | 2413 | ALA | PNA |
| 67 | Kannamal | 30 | F | 2239 | HL | Conservative |

| | | | | | | |
|---|---|---|---|---|---|---|
| 68 | Mabasha | 27 | M | 1481 | ALA | PNA |
| 69 | Palanisamy | 45 | M | 43957 | HCL | Marsupialization |
| 70 | Balasubramani | 32 | M | 59831 | ALA | PNA |
| 71 | Maran | 38 | M | 4587 | PLA | PNA |
| 72 | Palanisamy | 26 | M | 3239 | ALA | PNA |
| 73 | Ramu | 29 | M | 4270 | ALA | PNA |
| 74 | Gopalakrishnan | 27 | M | 5362 | ALA | CT |
| 75 | Karupan | 40 | M | 3249 | ALA | PNA |

**ALA** – Amoebic Liver Abscess

**PLA** – Pyogenic Liver Abscess

**HCL** – Hydatid Cyst Liver

**SCL** - Simple Cyst Liver

**HL** - Haemangioma Liver

**CT** - Chemo Therapy

**PNA** – Percutaneous Needle Aspiration

**EL** – Emergency Laparotomy

**ICD** – Inter Costal Drainage

**PC&TD** - . Partial cystectomy & tube drainage

**PC&C** - Partial cystectomy & capitonnage

**P&A C** – Pleural & Abdominal Cavity.